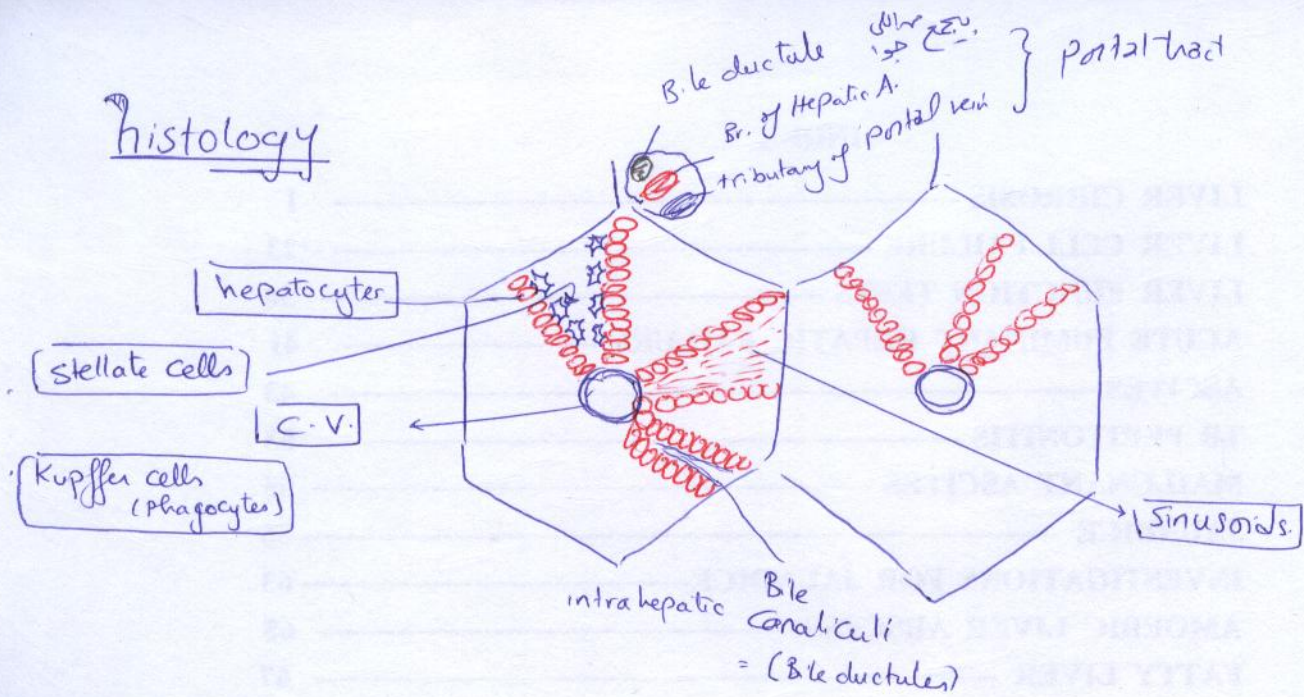


histology



Extrahepatic Bile ducts
 Hepatic ducts
 CHD
 C.O.
 CBD

← لیسے خارج لیسے لیسے

diffuse



لیسے لیسے لیسے
 - fatty liver
 - cirrhosis

focal



لیسے لیسے لیسے
 tumor

VISIT...

LANZAROTE
Caliente.COM

LIVER CIRRHOSIS

نظري SQ, LQ
Clinical

أكثر SQ

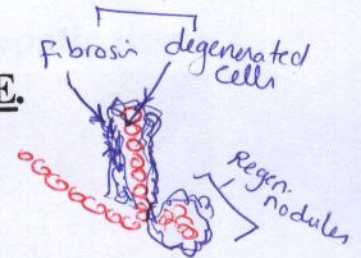
DEFINITION

البنية تأخذها أي حبة
not

- **DIFFUSE** Chronic liver disease characterized by:

1. **Degeneration.**
2. **Fibrosis.**
3. **Regeneration nodules.**
4. **Loss of normal structure:**

IRREVERSIBLE.



PATHOGENESIS

Q: + أكتة عده
gh'o

1. Hepatocellular necrosis leads to damage of the hepatic parenchyma & **Degeneration.**
2. Excessive **Fibrosis** occurs due to excessive proliferation of connective tissue, why?
Injury of hepatic parenchyma → **activation** of stellate cell → +++ secretion of **TGF-β₁** (Collagen fibres)
3. **Regeneration nodules:** will be formed. *damaged liver cells surrounded by fibrosis*
4. **Loss of the normal hepatic structure:** **IRREVERSIBLE.**

The liver may be initially **enlarged**, but with progression of the disease, it **shrinks**

5. COMPLICATIONS:

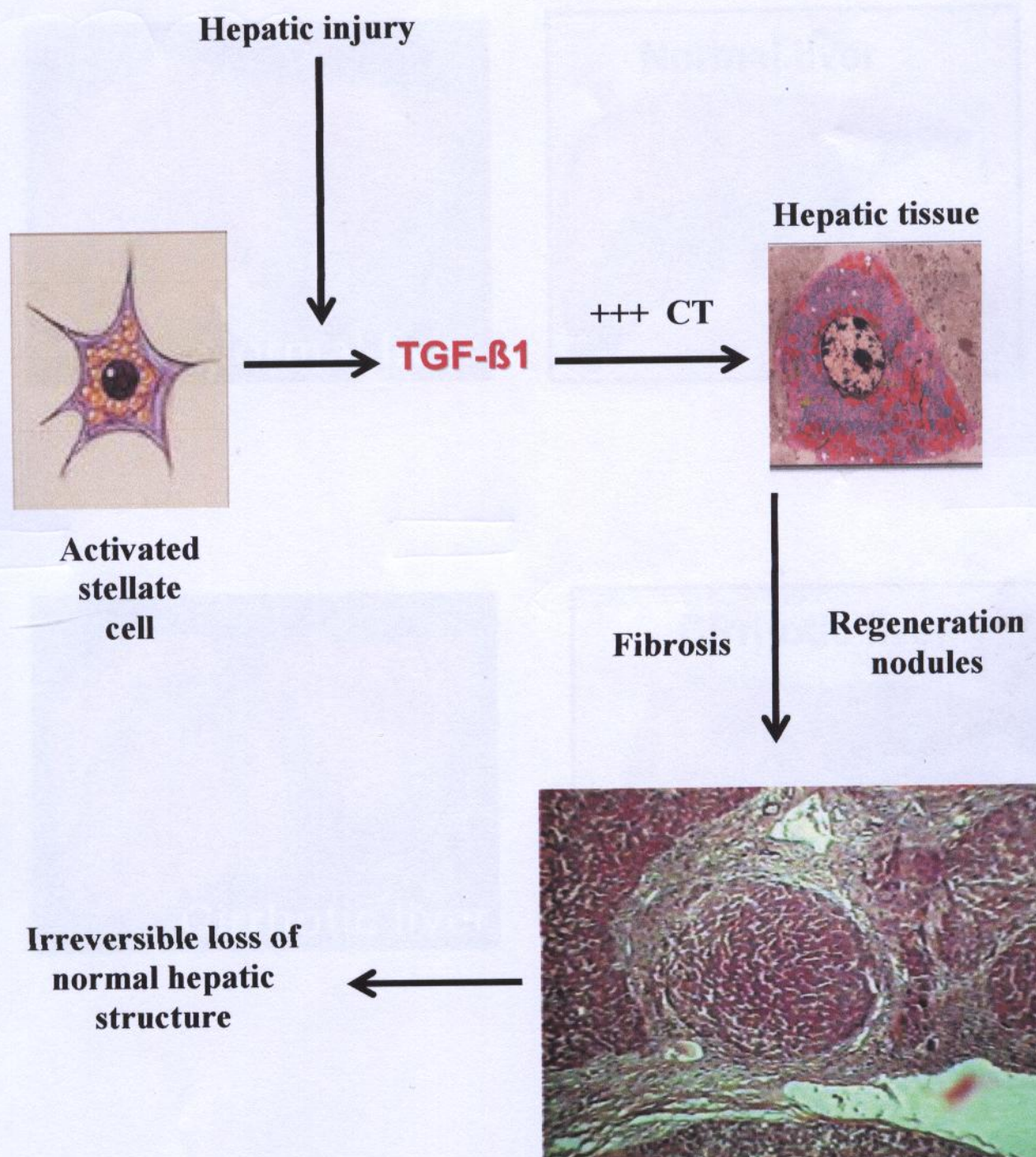
- Liver cell failure: due to progressive deterioration of liver functions.
- Portal HTN: the fibrous septa will block the portal flow of blood through the liver. *تضيق*
- HCC: may occur.

in advanced cirrhosis
excessive fibrosis in
(↓ liver span)

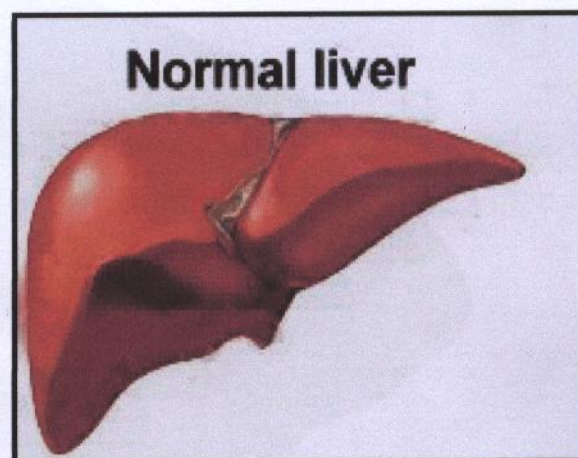
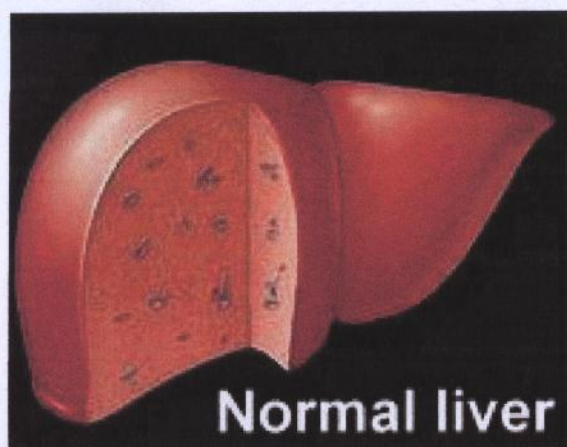
CLASSIFICATION

1. Morphological classification: **micronodular**, **macronodular**, **mixed**.
2. Etiological classification: see the **etiology**.
3. Functional classification: **compensated** & **decompensated cirrhosis**.
4. Child's classification: for grading the **severity of cirrhosis**.
حدوا فالقوا و عيالقوا

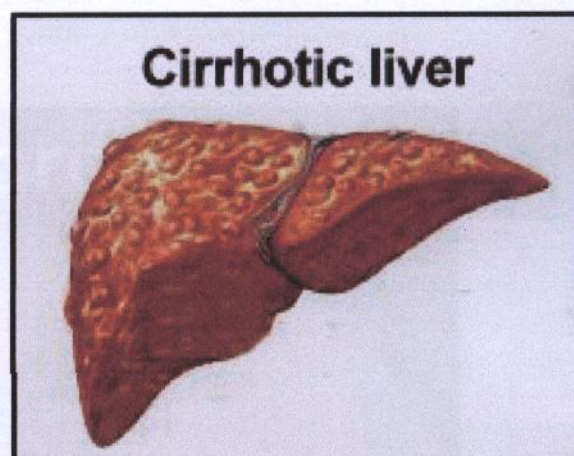
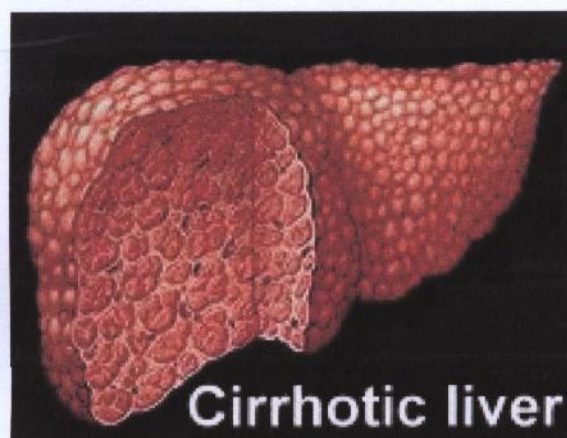
PATHOGENESIS OF LIVER CIRRHOSIS



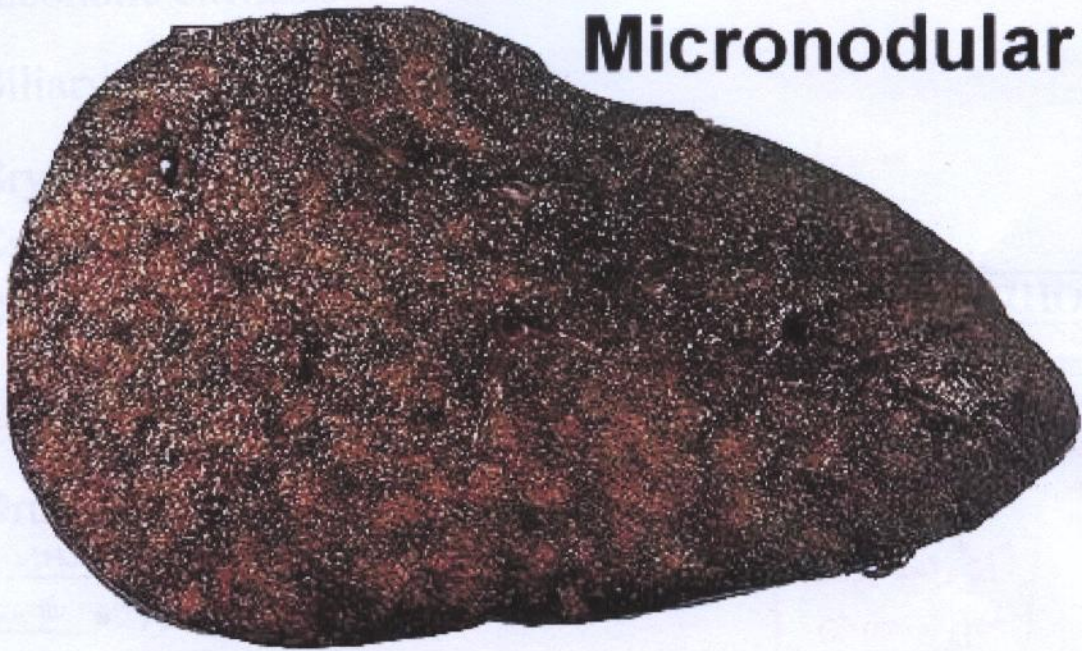
Micronodular



CIRRHOSIS

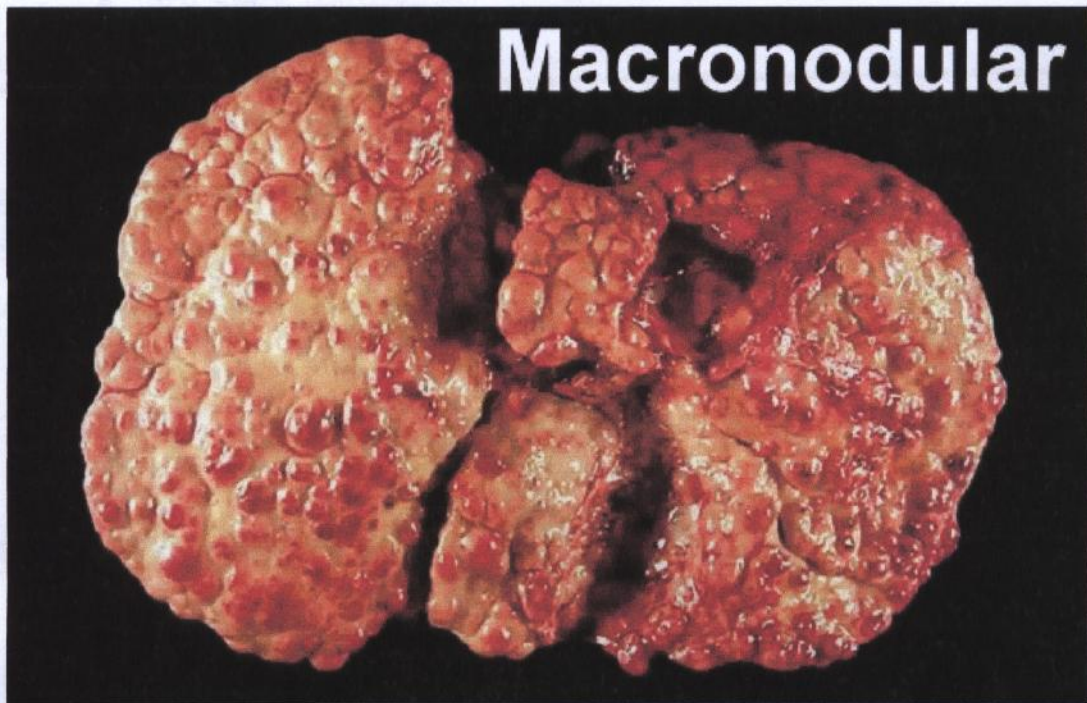


Micronodular



CIRRHOSIS

Macronodular



All cause → injury

ETIOLOGY

A B C D

1. **Alcoholic cirrhosis.** → most common cause of cirrhosis in America Europe
2. **Biliary cirrhosis.** [مخوش بیل]
3. **Cryptogenic (Idiopathic) cirrhosis.** by exclusion
4. **Cardiac cirrhosis.** [مخوش دما]
5. **Chronic hepatitis:**

POST-HEPATITIS CIRRHOIS

(Virus: B, C, D).
(Lupoid hepatitis).

A, E
are only
Acute
not chronic

لازم
(لو acute آسیب)
(cirrhosis لا یجمل)

- Postviral hepatitis
- Autoimmune

6. Drug-induced cirrhosis:

- Alpha methyl dopa. anti HTN
- Amiodarone. class III
- INH. anti TB
- Methotrexate. Immuno suppressive (Cytotoxic) → autoimmune Cancer

Oral
[p]

اکثر واحد مزمن
[chronic یجمل]

7. MISCELLANEOUS:

- Metabolic causes.
- NASH. = Non Alcoholic Steato Hepatitis

N.B.
Paracetamol is v. toxic on liver but not usually cause cirrhosis
نر وارد یجمل
Acute liver failure

Hepatic schistosomiasis

- There is ONLY fibrosis. only periportal
- There is No true cirrhosis: No degeneration, No regeneration nodules, No loss of structure of liver cells



CLINICAL PICTURE

A. Compensated "Latent" cirrhosis:

[EARLY]

- ASYMPTOMATIC: Early, there is no impairment of liver functions.
- ACCIDENTAL DISCOVERY: of an enlarged liver & maybe an enlarged spleen:
OR:

- During routine check up,
- During examination for a non-related condition.

OR:

RES
hyperplasia

[Porta HTN] ده متاخر اوری
spleen کبر تر عیال Liver یقی بسبب
نکه اعدادی انه یکبر کدا کجروم RES وای یومو قف Liver
WhiteKnightLove

B. Decompensated "Manifest" cirrhosis: [LATE]

I. General features of cirrhosis:

1. ABDOMINAL EXAMINATION:

- Liver: **Shrunk**, **Sharp edge**, Firm consistency.
- Spleen: Enlarged. *Compensatory RES*

2. FEATURES OF COMPLICATIONS:

- Liver cell failure. *eg: jaundice, ascites, bleeding tendency, fetor hepaticus*
- Portal HTN. *eg: hematemesis (esophageal varices) & Caput medusae*
- HCC. *eg: mass in Rt hypochondrium*

II. Specific features of cirrhosis:

- Features according to the specific cause of cirrhosis: "see later".

INVESTIGATIONS

I. General investigations of cirrhosis:

1. ABDOMINAL EXAMINATION:

- Liver imaging: Abdominal ultrasonography & Abdominal CT scan.
- Liver biopsy. *Definitive → 1, 2, 3, 4*

2. FEATURES OF COMPLICATIONS:

- Liver function tests: for (Albumin & prothrombin) Liver cell failure.
- Investigations: for (upper endoscopy to detect varices) Portal HTN.
- Alpha fetoprotein: for early detection of HCC.

II. Specific investigations of cirrhosis:

- For detection of the specific cause of cirrhosis: "see later".

CHILD'S CLASSIFICATION

- The severity of liver cirrhosis can be assessed using CHILD'S CLASSIFICATION:

Class	A	B	C
Encephalopathy	None	Minimal	Advanced coma
Ascites	None	Easily controlled	Poorly controlled
Serum albumin (gm / dl)	Over 3.5	3 - 3.5	Below 3
Serum bilirubin (mg / dl)	Below 2	2 - 3	Over 3
INR	Below 1.7	1.7 - 2.3	Over 2.3

TREATMENT

I. General TTT of cirrhosis:

1. TTT of:
2. TTT of:
3. TTT of:

Liver cell failure.

Portal HTN.

HCC. *surgery chemotherapy*

Complications

4. Antifibrotic drugs: (e.g. Colchicine, Corticosteroids) are of doubtful value. *(no)*

5. **LIVER TRANSPLANTATION:** the only curative ttt. *as irreversible loss of structure*

II. Specific TTT of cirrhosis:

- TTT of the specific cause of cirrhosis: "see later".

to avoid
Collagen
deposition
و تجنب
الليفات
الكبدية
تأكل
الأنسجة

ALCOHOLIC CIRRHOSIS

SQ *دخري*

ETIOLOGY

*لازم نذكر سبباته
toxic*

- Prolonged consumption of alcohol → toxicity to the liver cells.

PATHOLOGY

- General features of cirrhosis (usually Micronodular type).

- Liver cells may show:

- Eosinophil deposits.
- Fatty infiltration

*noteosinophil cells but eosinophilic granules
intra cytoplasmic*

(STEATOSIS).

Alcohol inhibits

*Lipophilic
factors
(apo lipoproteins)*

*Alcohol inhibits
Lipophilic factors (apo lipoproteins) to wash (sequester) fat from liver cells to blood*

*(Mallory bodies)
become red
by H&E
but not specific for Alcoholic Cirrhosis*

CLINICAL PICTURE

1. General picture of cirrhosis.
2. History of prolonged alcohol intake.
3. Associated complications of:

Alcoholism.

*eg:
كل عنوانه الخمس
المرتين
وعلى كل واحد
20g*

WhiteKnightLove

very non specific

Complications of Alcoholism

1. Cardiac:

- Coronary atherosclerosis. ($\downarrow$ risk)
- Dilated cardiomyopathy.

2. Neurological:

- Cerebral atherosclerosis. ($\downarrow$ risk)
- Myopathy. ery
- PN.
- Ataxia.
- Headache & migraine.
- Wernick's encephalopathy.
- Coma.
- Epilepsy. $\leftarrow$ Cause ppt factor (trigger)

3. Liver & GIT:

- Liver: Fatty liver, ^{chronic} hepatitis, cirrhosis.
 - GIT: gastritis, peptic ulcer, malabsorption, ^{Acute} pancreatitis.
- reversible irreversible fatal

4. BLOOD:

- Macrocytic anemia.

5. GENERAL:

- Metabolic: Hyperlipidemia, / Hyperuricemia, / Ketoacidosis.
- Parotid: Bilateral parotid enlargement.
- Palmar fascia: Dupuytren's contracture.

INVESTIGATIONS

1. General investigations for cirrhosis.

TREATMENT

1. General ttt of cirrhosis.

Gamma Glutamyl Transferase

2. GGT: Elevated.
v. non specific

2. Stop alcohol intake.

POST - HEPATITIS CIRRHOSIS

ETIOLOGY

- It follows chronic hepatitis due to:

- Postviral hepatitis
- Autoimmune

(Virus: B, C, D).
(Lupoid hepatitis).

not the only 2 causes
بني آدم جميع

PATHOLOGY

1. General features of cirrhosis.

2. Features of chronic hepatitis may be present.

3. Specific pathology of the cause: e.g.

- HBV: GGA & Positive orcin stain.
- HCV: Lymphocyte deposits & Fatty infiltration (STEATOSIS).

non-specific

CLINICAL PICTURE

1. General features of cirrhosis.
2. Specific past history of the cause: e.g.
" " of exposure

INVESTIGATIONS

1. General investigations of cirrhosis.
2. Specific investigations of the cause:

- Postviral hepatitis (Virus: B, C, D): HEPATITIS MARKERS.
- Autoimmune (Lupoid hepatitis): AUTO - ANTIBODIES.

TREATMENT

1. General ttt of cirrhosis.
2. Specific ttt of the cause:

VH & Lupoid hepatitis (see later).

antiviral

corticosteroid
immuno suppressive drug

BILIARY CIRRHOSIS

ETIOLOGY

- It is due to: [Long standing biliary obstruction = Cholestasis]

- Primary Biliary Cirrhosis (PBC) = Intra-hepatic biliary obstruction.
 • Secondary Biliary Cirrhosis = Intra or extra-hepatic biliary obstruction.

I. PRIMARY BILIARY CIRRHOSIS

ETIOLOGY

- Unknown: *probably autoimmune*
 • It is associated with other autoimmune diseases, e.g. SLE, Sjogren's syndrome.
 • Presence of anti-Mitochondrial antibodies in the serum, High serum IgM.

PATHOLOGY

1. General features of cirrhosis.
 2. Specific feature of PBC:

"Injury of the interlobular or septal bile ducts"

CLINICAL PICTURE

1. **INCIDENCE**: 90 % of the patients are females, 40 – 60 years.
 2. **Pruritus**: is the earliest symptom. (M → Y)
 3. **Jaundice**: obstructive jaundice occurs months to years after pruritus.
 4. **Steatorrhea & malabsorption of fat – soluble vitamins**:
 • **Bone affection**: osteoporosis & osteomalacia (Hepatic osteodystrophy)
 • **Bleeding tendency**:
 5. **Hepatomegaly**: enlarged, firm, with sharp edge.
 6. **Splenomegaly**: RES hyperplasia. retained bile Fibrosis
 7. **Skin hyperpigmentation & clubbing**. (autoimmune)
 8. **Associated autoimmune diseases** may be present, e.g. SLE, Sjogren's syndrome

non toxic non hypoxic

Liver enzymes

-↑ ALP
-↑ GGT
-↑ 5 NT
nucleotidase

from mucosa of Bile ductules & ducts
↑ markers of biliary obstruction

11

INVESTIGATIONS

1. Liver function tests:

[Obstructive jaundice], e.g.

- Serum alkaline phosphatase & GGT: elevated.
- Serum bilirubin (direct): elevated.

مستعار منه
نزلوا
فزيدوا في الدم
ويزيدوا في الدم
↑ indirect
hemolysis

2. Serology:

- Positive AntiMitochondrial antibodies: in more than 95 % of the patients.
- High serum IgM level.

3. Imaging:

[Abdominal ultrasonography or CT]

- Enlarged liver with: No dilated intra-hepatic biliary radicles.

U/S

4. Liver biopsy:

- Injury of the interlobular or septal bile ducts.

sensitive
good -ve test
لا يوجد موجودة تقريباً 100%
لو قفتم -ve - اندرأ صغير المرض
لو قفتم +ve
مقدري
أحطت لانا
في أمراض
أخرى
- SLE
- lipid hepatitis
↓
not specific

TREATMENT

I. General TTT of cirrhosis:

- LIVER TRANSPLANTATION: the only curative ttt.

II. Specific TTT (Symptomatic):

- Cholestyramine: Bile salt sequestrant (↓ pruritus).
- Ursodeoxycholic acid ↑ bile flow through the liver (↓ cholestasis).
- FAT - SOLUBLE VITAMINS (Parentrally).

Oral? Bile flow يزيد
Ursodeoxycholic acid

حسكي
ويطرد

cholagogue

II. SECONDARY BILIARY CIRRHOSIS

ETIOLOGY

1. Extra-hepatic biliary obstruction:

Extra-hepatic cholestasis

- Obstruction: in LARGE bile ducts that lie OUTSIDE the liver (CBD or its main branches).
- Causes: see obstructive jaundice. eg Gall stone, stricture

2. Intra-hepatic biliary obstruction:

Intra-hepatic cholestasis

- Obstruction: in SMALL bile ducts that lie WITHIN the liver.
- Causes: see obstructive jaundice. eg viral hepatitis, OCP, chlorpromazine

حسكي
side effects
Bile flow → OCPs, chlorpromazine

Cholestasis
يزيد
production of bile

CLINICAL PICTURE

1. Features of obstructive jaundice: of long duration.
2. Features of the cause of obstructive jaundice.
3. General features of cirrhosis: following longstanding obstructive jaundice.

INVESTIGATIONS

1. Liver function tests: [Obstructive jaundice], e.g.

- Serum alkaline phosphatase & GGT: elevated.
- Serum bilirubin (direct): elevated.

2. Investigations for the cause of obstructive jaundice: e.g.

- Imaging [Abdominal ultrasonography or CT]



1. Extra-hepatic biliary obstruction: Extra-hepatic cholestasis
- Enlarged liver with: dilated intra-hepatic biliary radicles.

2. Intra-hepatic biliary obstruction: Intra-hepatic cholestasis
- Enlarged liver with: No dilated intra-hepatic biliary radicles.

3. General investigations of cirrhosis.

TREATMENT

I. General TTT of cirrhosis:

(see before)

II. Symptomatic TTT:

- Cholestyramine: Bile salt sequestrant (↓ pruritus).
- Ursodeoxycholic acid ↑ bile flow through the liver (↓ cholestasis).
- FAT – SOLUBLE VITAMINS (Parentrally).

III. Specific TTT of the cause:

e.g.

- Surgical removal of gall stones.

ing medical
ing surgical

ing medical
ing surgical

White Knight Love

CARDIAC CIRRHOSIS

ETIOLOGY

الكبد باظ لأنه محتقن بالدم
"Congestive Hepatopathy"

السمنة
الألم
لحم متعب
القلب
هو محوش دما
السبب
الوحيد
له أهم سبب

- It is due to: [Long standing hepatic venous congestion]

1. Prolonged severe RVF.
2. Tricuspid valve disease: (TS, TR).
3. Prolonged severe pericardial disease (pericardial effusion, CP).
4. High IVC obstruction.
5. Budd-Chiari syndrome.
6. Veno-occlusive disease (VOD).

also in PNH thrombosis

- Long standing hepatic venous congestion will lead to:

GRF

1. ↓ drainage of sinusoidal blood flow into terminal hepatic venules → sinusoidal stasis.
2. Sinusoidal stasis → accumulation of deoxygenated blood, parenchymal atrophy, necrosis, collagen deposition, and, ultimately, fibrosis.
stellate cells

PATHOLOGY

1. General features of cirrhosis.
2. Centrilobular congestion.

also liver failure
↓
↑CO HF

CLINICAL PICTURE

1. General features of cirrhosis.
2. Liver: enlarged, firm,
3. Specific features of the cause, e.g.

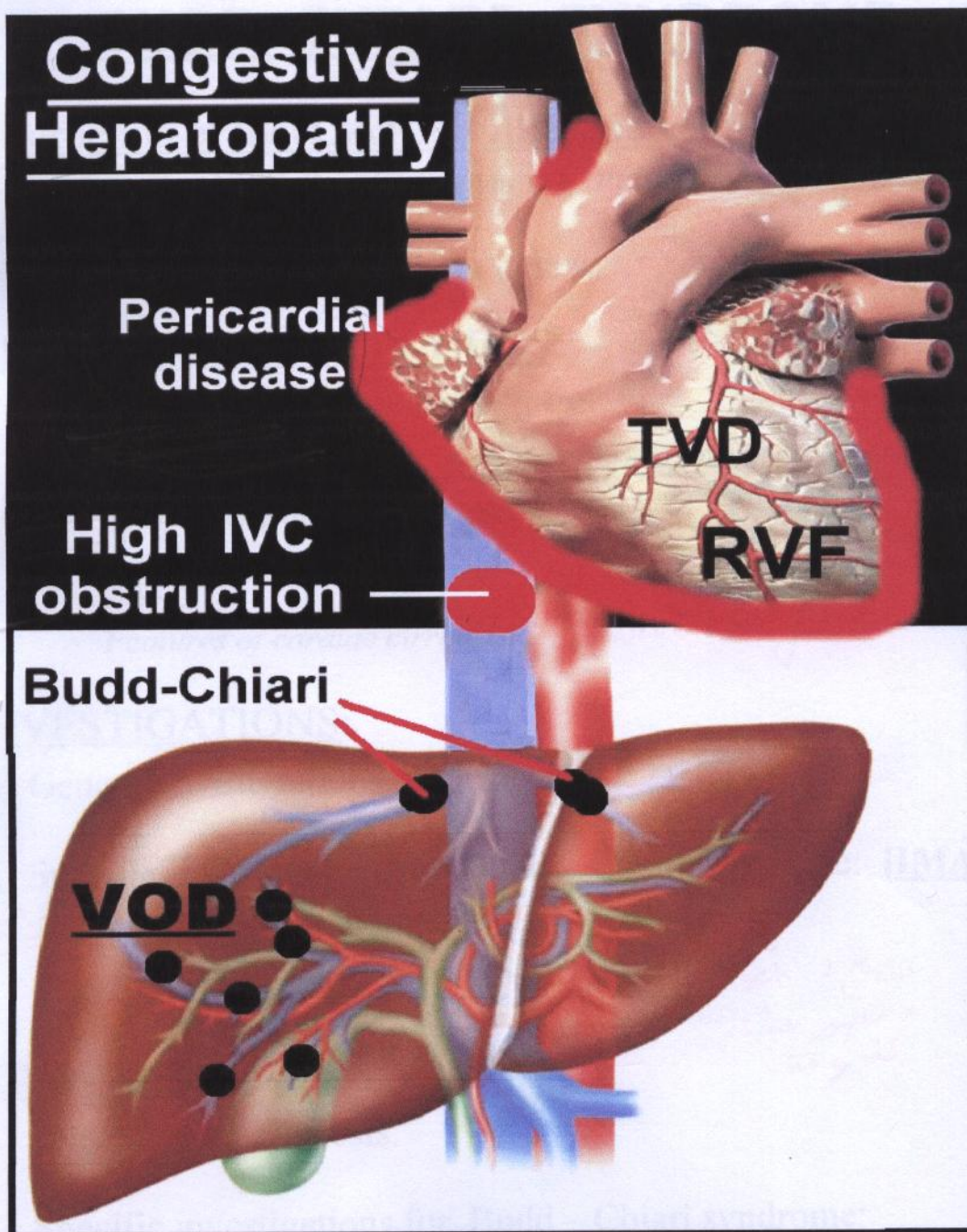
Causes of enlarged tender liver?
① Congested
② Inflamed
③ Malignant
④ fatty liver (rare to cause large & tender liver)
Stretchy Capsule
TENDER, with sharp edge.
RVF. [Congested neck vv. edema L-L.]
rubs

INVESTIGATIONS

1. General investigations of cirrhosis.
2. Liver imaging: [Abdominal ultrasonography & CT scan]
 - Enlarged congested liver.
 - Prominent hepatic veins. (dilated)
3. Specific investigations of the cause, e.g. cardiac imaging (echocardiography).

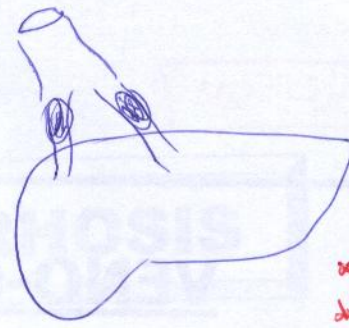
RV, Tricuspid, Pericardium

CARDIAC CIRRHOSIS



TREATMENT

1. General ttt of cirrhosis.
2. Specific ttt of the cause, e.g. RVF.



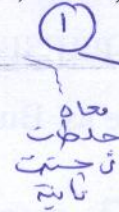
ازای OCP
؟ Curhosijsai
* zry biliary
* Budd & Chiari

BUDD-CHIARI SYNDROME

Case
بیماری، جاب

- Obstruction of the **LARGE** hepatic veins due to:

1. Thrombosis: Hypercoagulation (e.g. PRV, PNH, CCPs), Behcet's syndrome.
2. Invasion from outside: Malignancy.
3. No cause: In many cases (~ 50 %).



↓
Clotting factors
but causes
venulitis

- CLINICAL PRESENTATIONS:

1. Acute:

- Acute abdominal pain, hepatomegaly, ascites & mild jaundice.
- Acute liver failure may occur.

stretch capsule by congested liver



sudden portal HTN

2. Chronic:

- Features of cardiac cirrhosis.

chronic liver failure

↓
Bile canaliculi
partially
occluded
by
congested
liver

- INVESTIGATIONS:

1. General investigations of cirrhosis.

2. Specific investigations for Budd – Chiari syndrome: **[IMAGING]**

- MRV. Magnetic resonance venography
- Duplex ultrasound.

تصویر برداشتن
و تصویر گرفتن
تسویه

MRA
-TIA

- TREATMENT:

1. General ttt of cirrhosis.

2. Specific investigations for Budd – Chiari syndrome:

- Acute: Fibrinolytic therapy, followed by anticoagulants.
- Chronic: Anticoagulants, to prevent further thrombosis.

VENO-OCCLUSIVE DISEASE

- Obstruction of the **SMALL & MEDIUM-SIZED** hepatic veins due to:

1. Toxic injury by a toxin: Senecio alkaloid present in a special type of medicinal tea.
بعض الأعشاب من قبا في أفريقيا
2. Chemotherapy, Radiotherapy.
3. Following BMT. *in preparatory stage due to drugs: Busulfan*
injury of venules

- CLINICAL PRESENTATIONS:

"A disease of CHILDREN"

- The same as Budd – Chiari syndrome.

- INVESTIGATIONS:

- The same as Budd – Chiari syndrome.

- TREATMENT:

- The same as Budd – Chiari syndrome.

METABOLIC CIRRHOSIS

ETIOLOGY

1. Hemochromatosis
2. Wilson's disease
3. Alpha 1- antitrypsin deficiency.

"Iron overload".

"Copper overload".

1. HEMOCHROMATOSIS

"Bronze Diabetes"

hyperpigmentation
حیدر فی اکلر

حیدر فی اینتریکس

DEFINITION

- Iron storage disorder: Massive generalized iron deposits in various organs.
- Tissue damage: Functional impairment in various organs.

ETIOLOGY

1. PRIMARY:

- Unknown defect:

"HEREDITARY"

[Excessive intestinal iron absorption].

حلل فی
ferritin
apoferritin
system
الی
فی
small
intestine

الی بچکم
فی انتصاب اکلر
عزوبارط فکتم کبره نه اکلر روح اکلر

2. SECONDARY:

- a) Increased production: chronic hemolytic anemia especially Thalassemia.
- b) Decreased utilization: sideroblastic anemia.
مقویات عاده نه السوبر مارکت
- c) Increased intake:
 - ↑ parenteral intake: repeated iron injections & repeated blood transfusions.
 - ↑ dietary intake: medicinal tonics & red wine.

overload ← anaphylactic shock ← لو صحتش وکرها ←
عالمه ضعیفاً نه عیوه اقلاله ←
یاخذ حیدر کولاجن ←
overcorrection ← collection ←
اوعیه بجمع نقص الحیدر و اخذه بجمع collection

Polyarthritia (in 50% of patients)

Skin
Liver
Pancreas DM

Commonest cause of death here is HCC other causes (HF & LCF)

Commonest cause of death here is HCC other causes (HF & LCF)

18

CLINICAL PICTURE

"SHIP O"

- **S**ex: usually males between 40 – 60 years.
- **S**kin: hyperpigmentation (bronze coloration).
- **S**uprarenal: adrenal insufficiency. hypofunction

not common in ♀s as menstruation removes Fe load

- **H**epatic: liver cirrhosis → HCC (25%).

- **H**eat: cardiomyopathy.

- **I**nfections: uncommon. (v. uncommon)

Fe is considered as nourishment of some organisms (yersinia)

- **P**ancreas: secondary diabetes.

- **P**ituitary: hypogonadism (testicular atrophy, impotence, ↓ libido, sterility)

- **P**olyarthritia. deposited in synovial membrane

- **O**thers: mental disturbances. prefrontal

HCC (25%)

Hemochromatosis
Cirrhosis
HCC

DCH
RCH → HF

as Fe overload inhibits phagocytosis [as ↓ lymphocytes & macrophages]

↓FSH
& LH

testis atrophy

testis Fe deposition
very rare

INVESTIGATIONS

1. General investigations for cirrhosis.

2. Investigations suggesting IRON OVERLOAD:

a) Laboratory:

- Serum iron, ferritin, transferrin saturation: increased.
- TIBC: decreased.

b) Liver Imaging: "Abdominal CT & MRI"

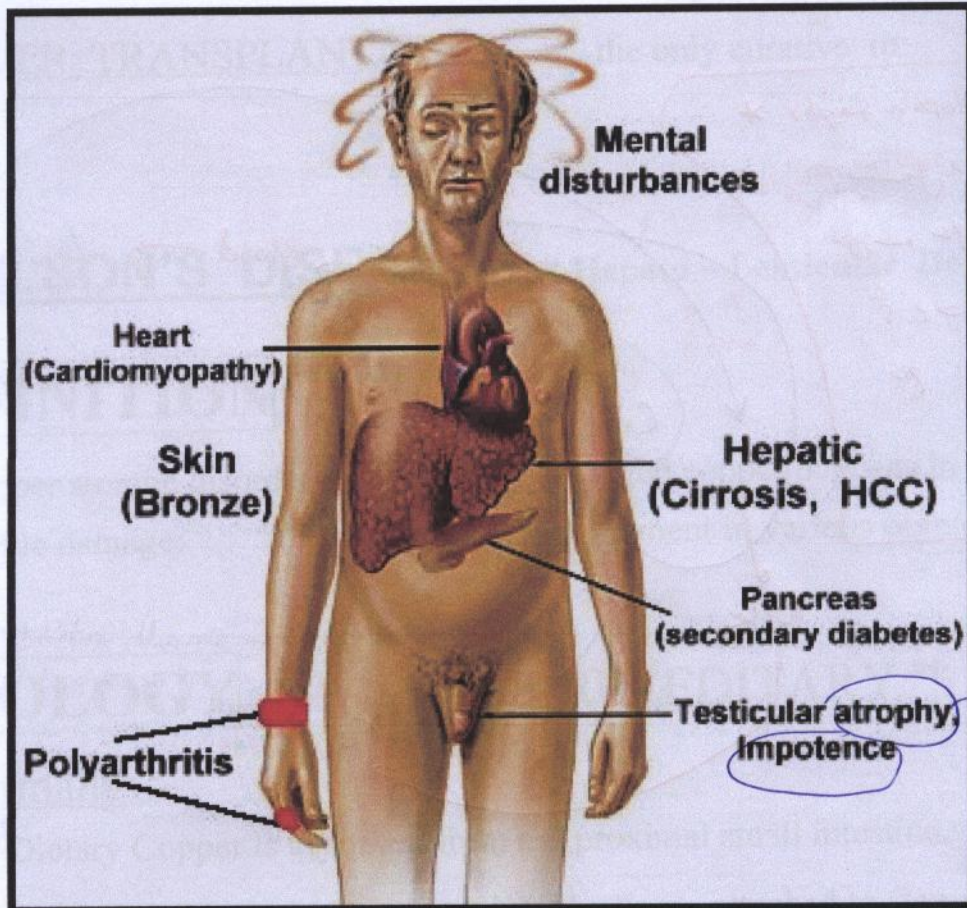
- Excess iron deposition in the liver.

c) Liver biopsy:

- Excess iron deposition in the liver. quantitatively
- Confirms cirrhosis. staining technique

HEMOCHROMATOSIS

"Bronze Diabetes"

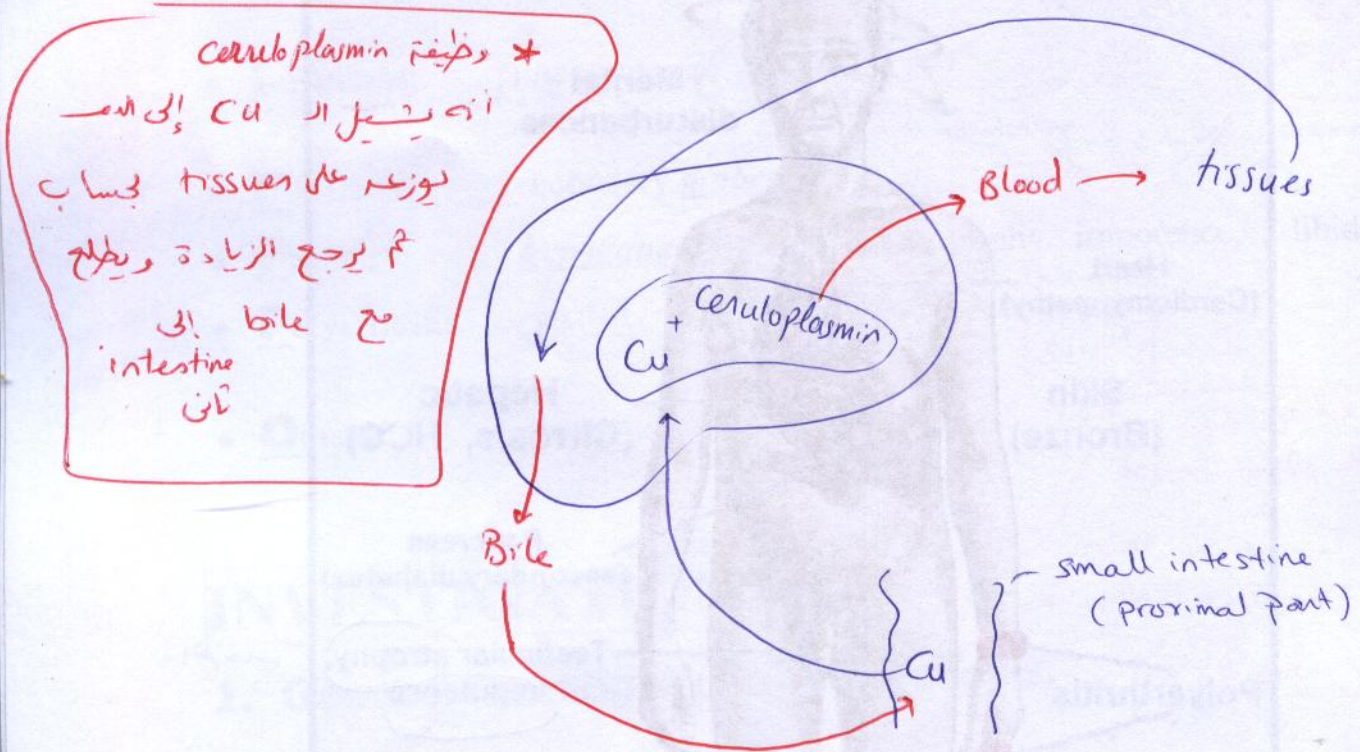


اهم حاسب

CLINICAL PICTURE

"S H P O"

HEMOCHROMATOSIS "Bronze Diabetes"



a) Laboratory:

- Serum iron, ferritin, transferrin saturation: increased
- TBC: decreased

b) Liver Imaging: Abdominal CT & MRI

- Excess iron deposition in the liver.

c) Liver biopsy:

- Excess iron deposition in the liver.
- Confirms cirrhosis.

TREATMENT

1. Restriction of iron in diet.

Fe def anemia
اشترج بکتاب

2. Removal of excess iron:

- o Iron chelation: DEFERRIOXAMINE increases urinary excretion of iron.
- o Repeated venesection.

3. Symptomatic treatment:

e.g. DM, HF.

adrenal insuff → cortisol

the only curative ttt.

4. LIVER TRANSPLANTATION:

insulin

Diuretics

high incidence of insulin resistance

MCC

liver
lentiform nucleus of B.G.

2. WILSON'S DISEASE

“Hepato – Lenticular Degeneration”

- H.A.
- Extra Δ

DEFINITION

- Copper storage disorder: Massive generalized copper deposits in various organs.
- Tissue damage: Functional impairment in various organs.

ETIOLOGY

“HEREDITARY”

- Normally:

- Dietary Copper is absorbed from the proximal small intestine.
- Absorbed Copper is then taken into the liver, attached to Ceruplasmin and released to the blood.
- Copper is excreted from the body through the BILE.

- In Wilson's disease:

- Mutation → decreased excretion of Copper through the bile →
→ Copper builds up in the liver and injures liver tissue.
- Deficiency of Ceruplasmin → Free Copper will be released to the blood →
Excess Copper will build up in blood & will be deposited in the tissues.

++ tissues
(biopsy)
++ in urine
(تحليل بول)

①
نیزل فی البول
ceruplasmin ↓

loosely attached to albumin

هو مش حر اوی له

If Blood measurement

Cu ↓

تissue urine

TREATMENT

1. Restriction of iron in diet.

اشترج بکتابه ~
Fe def anemia

2. Removal of excess iron:

- o Iron chelation: DEFERRIOXAMINE increases urinary excretion of iron.
- o Repeated venesection.

3. Symptomatic treatment:

e.g. DM, HF.

adrenal insuff → corticoid

4. LIVER TRANSPLANTATION:

the only curative ttt.

insulin

Diuretics

response ↓
high incidence
of insulin
resistance

MCC

liver
lentic form nucleus
of B.G.

2. WILSON'S DISEASE

“Hepato – Lenticular Degeneration”

- H.A.
- Extra Δ

DEFINITION

- Copper storage disorder: Massive generalized copper deposits in various organs.
- Tissue damage: Functional impairment in various organs.

ETIOLOGY

“HEREDITARY”

- Normally:

- Dietary Copper is absorbed from the proximal small intestine.
- Absorbed Copper is then taken into the liver, attached to Ceruplasmin and released to the blood.
- Copper is excreted from the body through the BILE.

- In Wilson's disease:

- Mutation → decreased excretion of Copper through the bile →
→ Copper builds up in the liver and injures liver tissue.
- Deficiency of Ceruplasmin → Free Copper will be released to the blood →
Excess Copper will build up in blood & will be deposited in the tissues.

++ tissues
(biopsy)
++ in urine
(تحليل بول)

١
نیزل فی البول
Ceruplasmin ↓

loosely
attached
to albumin

هو مش حر اوی که

If Blood measurement

Cu ↓

tissue
urine

WhiteKnightLove

CLINICAL PICTURE

- **Liver:** Cirrhosis, Hepatitis (chronic or acute).
- **Brain:**
 - Extrapyrarnidal disease (Parkinsonism or chorea).
 - Mental disturbances (Dementia).
- **Eye:** Kayser-Fleischer rings (green-brown rings).
- **Kidney:** Tubular affection (RTA). → around iris
- **Blood:** Hemolytic anemia.

lot
Hemo-
chromatosis
only
cirrhosis
& HCC

Renal
Tubular
Acidosis

INVESTIGATIONS

1. Investigations of Copper metabolism:

- Ceruplasmin: decreased in serum.
- Copper: decreased in serum & increased in urine.

MCQ : most common cause of cirrhosis that causes HCC?

1. Viral C > B
2. Hemochromatosis
3. Alcoholic

2. Liver biopsy:

- Excess copper deposition in the liver.
- Confirms cirrhosis.

TREATMENT

1. Restriction of copper in diet.

Avoid

Bean
Potatoes
leafy greens
liver, kidney
coffee

2. Removal of excess copper:

- o Copper chelation: PENICILLAMINE increases urinary excretion of iron.
- o K sulphide: precipitates copper in the intestine. → لذيذ لذيذ
أوخذ orally

3. Symptomatic treatment:

e.g. Parkinsonism.

eg: Cimet, L-dopa

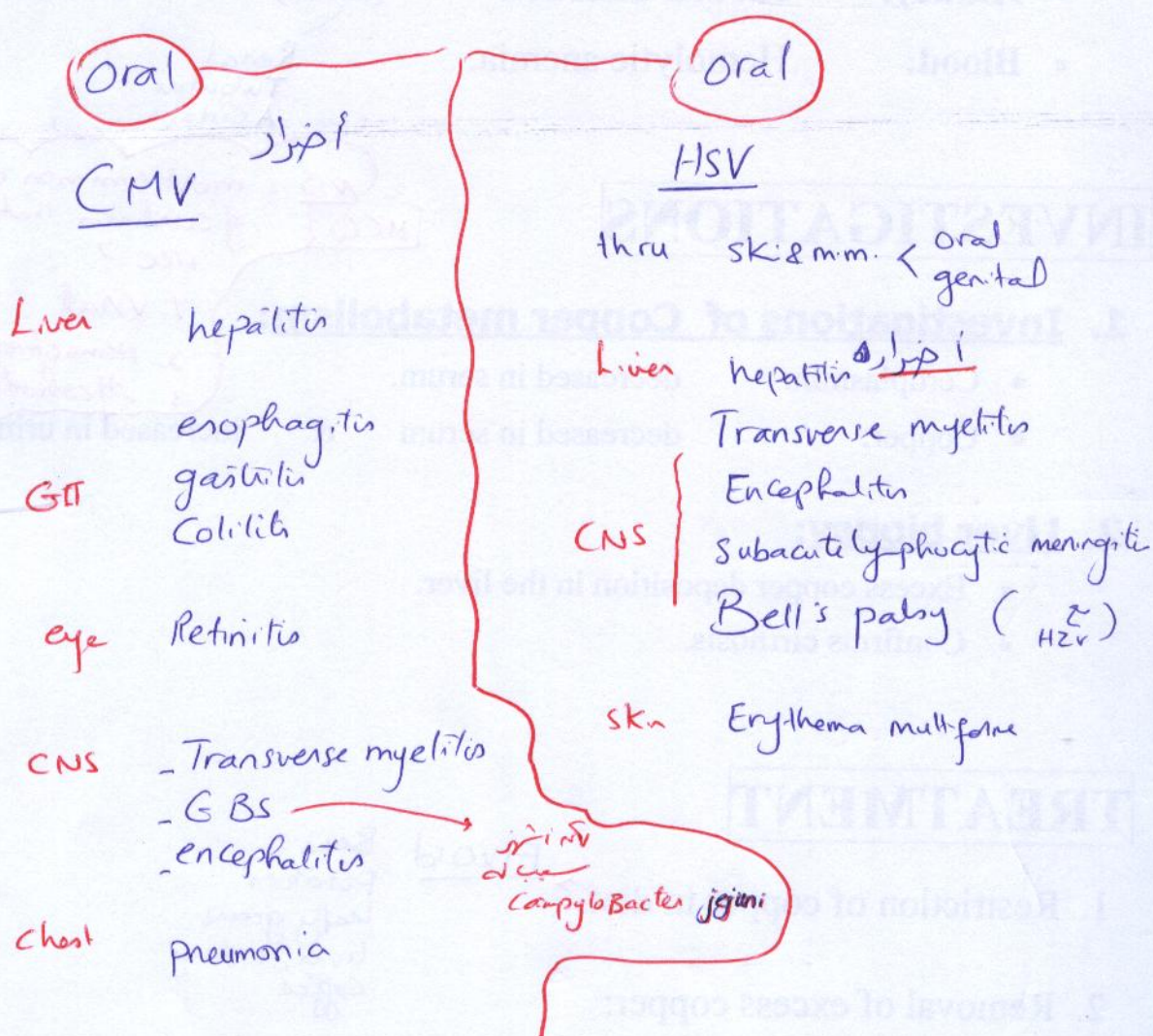
4. LIVER TRANSPLANTATION:

the only curative ttt.

3. ALPHA 1- ANTITRYPSIN DEFICIENCY

- A genetic disorder which presents with:

- Liver: liver cirrhosis, chronic hepatitis.
- Lung: emphysema (primary).



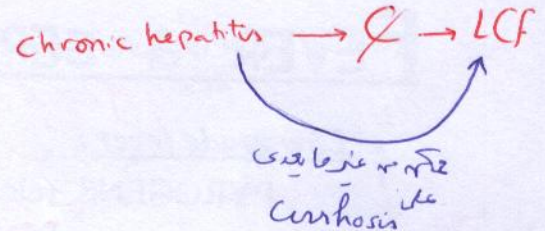
LIVER CELL FAILURE

ETIOLOGY

1. Advanced cases of:

- Liver cirrhosis. *Most common cause*

- Chronic active hepatitis.
- Obstructive jaundice.
- Hepatic malignancy.



2. Infections:

- Viral hepatitis:
- Other viruses causing hepatitis:

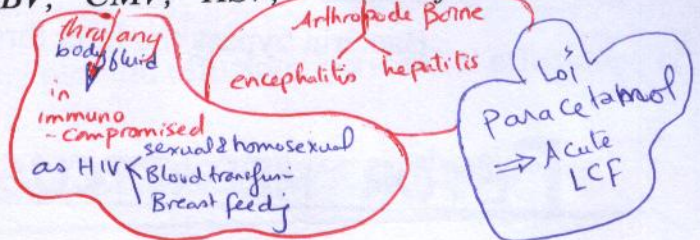
B, C, D.
EBV, CMV, HSV, Yellow fever.

3. Iatrogenic & (Toxins):

- Alcohol.
- Halothane.
- INH.

- (Carbon tetrachloride, DDT, Phosphorus.) ⇒ OPC organophosphorus compounds

Oral
injection



pesticide

CLINICAL MANIFESTATIONS

1. Failure of health.
2. Fever & septicemia.
3. Feter hepaticus.
4. Jaundice.
5. Cardiovascular manifestations
6. Ascites.
7. Skin manifestations.
8. Hematological manifestations.
9. Hepatic encephalopathy.
10. Endocrinal manifestations
11. Disturbed carbohydrate metabolism.
12. Hepato-renal syndrome.
13. Increased incidence of infections.

CLINICAL MANIFESTATIONS

FAILURE OF HEALTH

- Weakness, Weight loss.

FEVER & SEPTICEMIA

- Low grade fever:

PYROGENS released due to necrosis of the liver cells.
non specific

- Septicemia:

Failure of the liver to clear bacteria from the circulation,
Bacteria bypass the liver through port - systemic shunts.

OR:

FETOR HEPATICUS

- Sweetish, fecal smell:

Failure of detoxification of mercaptans absorbed from the intestine due to:
liver failure or shunts.

JAUNDICE

- Hepatocellular jaundice:

Failure of the liver cells to metabolize bilirubin.

CARDIOVASCULAR MANIFESTATIONS

1. Hyperkinetic circulation:

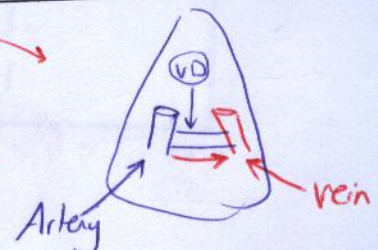
- Overproduction & decreased destruction of: vasodilator materials.

[Adenosine, $TNF\alpha$, NO]

2. Central cyanosis & clubbing:

- Pulmonary AV shunts:
- Porto-pulmonary shunts.
- Basal lung collapse due to tense ascites.

due to vasodilator materials.



due to portal HTN
pulmonary
venules
(دم زرد)
فقط
portal
venules
(دم زرد)

White Knight Love

① Liver failure to detoxify toxins of bacteria
② Bacterial toxins bypass liver thru portosystemic shunts (of portal HTN which is commonly caused by Cirrhosis which is the commonest cause of LCF)

تفسيرها

1. anorexia & ↓ intake (due to septicemia & toxemia)
2. malabsorption
3. ↓ ptn synthesis

why?
GRF

التيان يفسد وانه كان
وزنه يزداد فيه
ascites
بس هو يفسد

Page 24
Sulphur
containing
pus

كلام شفوي على أعلى مستوى

لو واحد عنده
فخف وياخذ
دوا الفخف
لو حاله
LCF
احتياجه لدواء
الفخف يقل
بسبب
VD materials

H.F
↑↓
LCF

Cardiac
Cirrhosis

GRF

ASCITES

- Incidence:

It is common in liver failure secondary to liver cirrhosis.

- Onset:

1) Gradual: the usual presentation in liver cirrhosis (common).

2) Acute: ascites may develop rapidly if the liver function is $\downarrow\downarrow$ acutely:

- Shock. *severe tissue hypoperfusion*
- Hemorrhage. $\rightarrow$ *Bleeding varices (hematemesis)*
- Infection. $\rightarrow$ *over stress on already exhausted liver*
- Portal vein thrombosis.

- Course:

It may be followed by: oedema LLs, pleural effusion, pericardial effusion.

- Pathogenesis: "Pathogenesis of ascites in liver cirrhosis"

1. **Hypoalbuminemia:** Due to diminished synthesis of **albumin** by the liver.
 "The ascitic threshold" for albumin is: 3 gm %.
 normal value 4-5 g-% $\rightarrow$ *لو اقل من 3 بيانيق*
2. **Portal Hypertension:** It rarely produces ascites. *الوحده*
 It localizes the transudate to the peritoneal cavity.
3. **Salt & water retention:** "Through stimulation of the **RAAS**"
 • **Overflow Theory:** salt retention is **primary** & then it starts the development of ascites.
 • **Underfill Theory:** salt retention is **secondary** to ascites & then it aggravates ascites.
4. **Lymphorrhea:** In post-sinusoidal obstruction, there is dilatation of the lymphatics on the surface of the liver with extravasation of lymph into the peritoneal cavity. *ascites exudate*
5. **Complications:** Development of TB peritonitis, malignancy or SBP.

Most recent theory

"The peripheral arterial VD theory"

- Cirrhosis $\rightarrow$ arterial VD $\rightarrow$ $\downarrow$ splanchnic & systemic vascular resistance $\rightarrow$
 $\rightarrow$ pooling of blood in the splanchnic circulation $\rightarrow$ $\downarrow$ effective AB volume $\rightarrow$
 $\rightarrow$ stimulation of the **RAAS** $\rightarrow$ salt retention $\rightarrow$ ascites.

albumin $\rightarrow$? $\rightarrow$ $\downarrow$ $\rightarrow$ salt & water

MCO

Most common skin manifest of LCF in spider naevi

SKIN MANIFESTATIONS

1. Arterial spiders "Spider naevi": due to estrogen excess

- They consist of a central dilated arteriole with radiating capillaries.
- Compression of the central arteriole, causes blanching of the whole lesion.
- They are found in the distribution of the SVC (face, neck, UL & upper chest).

clinical لا تظهر في

2. Palmar erythema:

due to estrogen excess

- The hands are warm. ← VD
- The palms are bright red in colour, especially the thenar & hypothenar eminences, and the heads of the metacarpal bones.

3. Paper-money skin:

عروق زرقاء رفيعة

due to estrogen excess

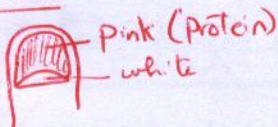
- Numerous small vessels scattered through the skin in random fashion.

4. White nails:

due to hypoproteinemia

- Opacity of the nail bed with a pink zone at the tip of the nail.

Normal



LCF



Causes of Palmar Erythema

1. Liver cirrhosis & Liver cell failure.
2. Leukemia. → Hairy cell leukemia: angiogenic mediators
3. Thyrotoxicosis. VD
4. Rheumatoid arthritis.
5. Pregnancy. ++ estrogen → VD
6. Familial.
7. OCPs (drug induced) ← OCP → ++ estrogen
Cortisol → Polycythemia
& Cushing

GFR

100% mortality

HEPATO-RENAL SYNDROME

- Renal failure with NORMAL renal histology occurring in patients with severe liver disease.

- Etiology:

↓ GFR

- Decreased effective plasma flow due to: systemic VD, ascites, diuretics.
- VC of the afferent renal arterioles due to: ↑ Vasopressin.

- Precipitating factors: excessive diuresis, diarrhea, vomiting, infection, tapping of ascites.

- Prognosis: it carries a very bad prognosis & is usually fatal.

- Treatment: avoid: the precipitating factors.

give: IV albumin (plasma expander),
Octreotide (causes systemic VC).

Synthetic Somatostatin
Sandostatin

pooling
ولا يقل GFR

plus,

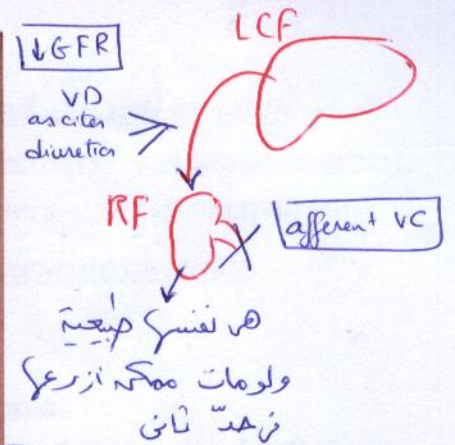
ما يجس نتيجه هبوط

يخفض الدم ثباته الى B.v.

السوية الى أنت حبيب
لأرجوا يسهطوا ثباته م. الدم

WhiteKnightLove

Spider naevi



(MCQ) Can be transplanted in others

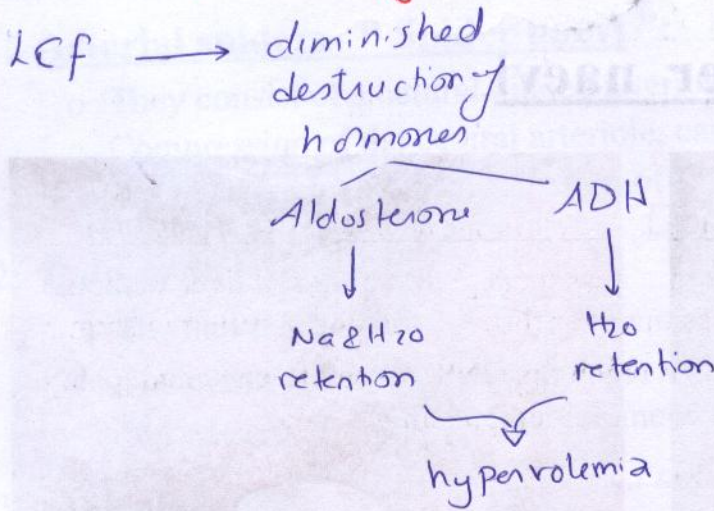
Palmar erythema



Salt & H₂O retention

1. Over flow theory:

→ Asites من الزيد



= Overfilled Circulation
++ hydrostatic pressure

→ Asites من الزيد

overflow in interstitium

Asites

من الزيد

Asites من الزيد

2. Under filled theory

من الزيد

Asites من الزيد

already موجود

Asites

(fluid exit from inside to outside B.V.)

= ↓ fluid inside B.V.

(underfilled)

(hypovolemia)

Kidney

++ RAAS

Na & H₂O retention

Most recently

Liver cell failure

→ VD of splanenic

→ Pooling

= visceral
mesenteric splenic hepatic

↓ Effective volume
Kidney

RAAS

HEPATIC ENCEPHALOPATHY

- It is a **neuropsychiatric syndrome** that may complicate severe liver disease.

- **Pathogenesis:** *ليه لا الابد تعب الخ تعب؟*

→ Q:

يكتب مع
ppting
factors

1. **Production of toxic substances:** *by the action of BACTERIA on intestinal proteins*

- These toxic substances will either pass through the diseased liver without detoxification ¹ or will bypass the liver through the porto-systemic shunts. ²

- These toxic substances will then reach the CNS & lead to encephalopathy because they will disturb the cerebral metabolism.

- These toxic substances include:

1 • Ammonia: It interferes with Krebs's cycle → ↓ energy supply to brain.

2 • Amines: Mercaptans, Serotonin. *(inhibitory on brain)*

2. **Disturbance of amino acids:**

a) *Increased aromatic aa* & *decreased branched aa* will result in:

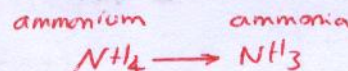
• Decreased production of normal neurotransmitters (as noradrenaline).

• Increased production of false neurotransmitters (as octapamine). *مائل*

b) *Increased level of GABA which is an inhibitory neurotransmitter.*

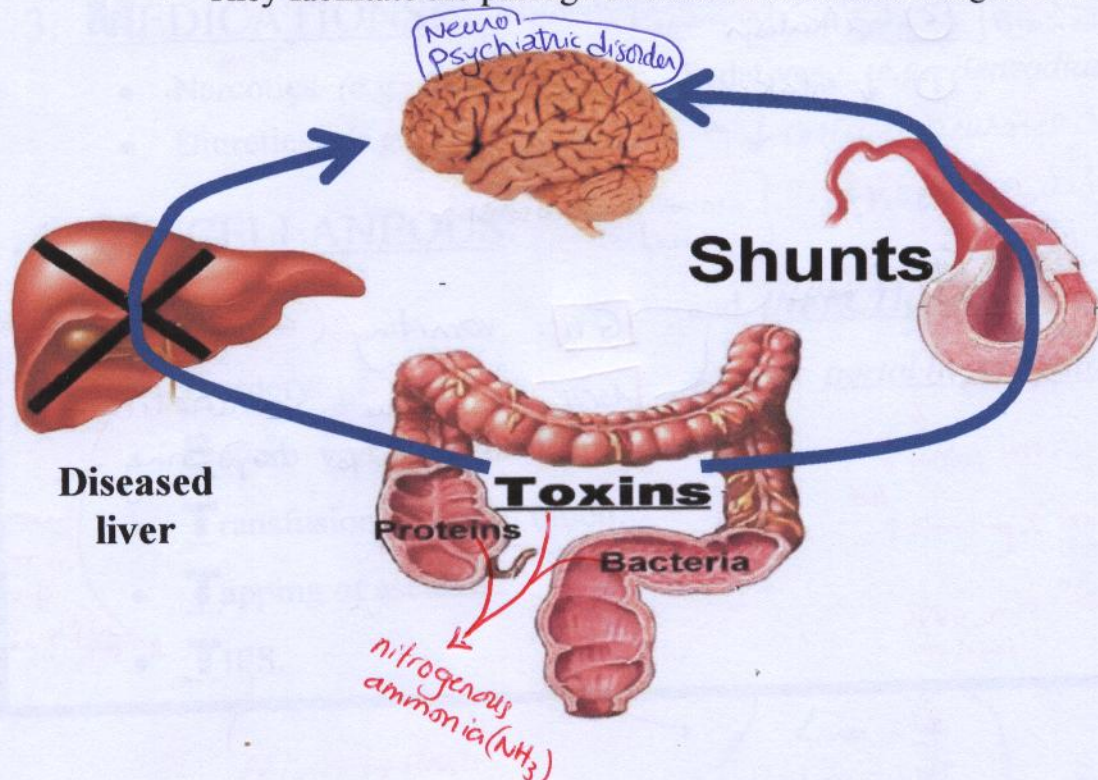
neuro-
transmission
is bad

3. **Alkalosis & Hypokalemia:**



• They increase the renal production of ammonia.

• They facilitate the passage of toxic substances through the BBB.



why
Alkalosis $\text{pH} \uparrow$:

① $\uparrow$ intake of alkali :

as pt is Liver failure
 $\downarrow$ ($\downarrow$ destruction of gastrin)
peptic ulcer or dyspepsia
 $\downarrow$

$\uparrow$ intake of Antacids

② $\uparrow$ loss of acid :

repeated vomiting (due to Congestive due to
Gastropathy PHTN)
repeated gastric drainage (due to hematemesis)

③ $\downarrow$ K

④ Thiazide : $\rightarrow$ side effect is Hypochloremic alkalosis

⑤ $\uparrow$ aldosterone

$\uparrow \text{Na}$
 $\downarrow \text{K}$
 $\downarrow \text{H}^+$ $\rightarrow \uparrow \text{pH}$

why $\downarrow$ K

① Aldosterone

② alkalosis

③ $\downarrow$ intake - anorexia
- dyspepsia
- malabsorption

④ $\uparrow$ loss

GI: vomiting (Congestive gastropathy)

Ascites $\rightarrow$ due to diuretics

due to Tapping

کما ناسی
تقومه
B.V.
و یفقد K اکثر

GRF → منكم
بشرة

- Important question: "Self - assessment"

← "Give reasons for: Alkalosis & Hypokalemia in liver cell failure".

PRECIPITATING FACTORS

"4 M"

1. **M**uch NITROGEN load:

- Excess dietary protein.
- Constipation. → also diarrhea
- Azotemia.
- GIT bleeding (e.g. Hematemesis):

عندى استرجاع على
العنب فقلنا في عيانية
دخلوا في coma لأنهم
كلوا بروتين في الزنا يوم الجمعة

كتب مع سؤال
Pathogenesis

أنا غير بس أفك
الإسهال من
أخلى إسرائيل

- It ↑ intestinal toxins due to action of bacteria on blood proteins.
- It ↓ the hepatic blood flow & thus worsens liver functions.
- It ↓ the renal blood flow & thus worsens renal functions.

↓ clearance
urea
creatinine
Nitrogen
anemia

المرضى
على
المرضى
prognosis
v. bad

2. **M**ETABOLIC disturbances:

- Alkalosis.
- Hypokalemia.
- Hyponatremia. (due to vomiting & diarrhea)
- Hypovolemia. (due to Bleeding)

التفسير
1. hypoperfusion
2. ↓ Na outside Brain cells → Fluid shifts from out to inside

3. **M**EDICATIONS:

- Narcotics (e.g. Morphine), absolutely Contra indicated
- Diuretics (e.g. Frusemide), Sedatives, (e.g. Benzodiazepines).

هو ال Na
يقرب على
بالنسبة لمرضى
Brain edema

4. **M**ISCELLANEOUS:

- S**BP,
- S**urgery:
- S**evere vomiting or diarrhea.
- T**ransfusion of stored blood.
- T**apping of ascites.

and INFECTIONS in general.

e.g. for portal hypertension.

↓ Portal HTN
but
↑ shunting (toxins reaches Brain أسرع)
وأيضا حالة "تعبير stress on liver"

Stress → **T**IPS.

shunts by stents
inside liver

liver ن
تلاتة بحركة
flapping tremor

encephalopathy
Tapping
منع مغناطيس تفل

- Clinical Picture:

2 stages

1. Precoma: ^{تأه}

S A D

- **S**leep disturbances: ^{late} somnolence & ^{early} inverted sleep rhythm. ^{نیام برعکس می باشد}
- **S**peech disturbances: slurred speech or monotonous speech. ^{pyramidal}
- **A**pathy. = poor cerebration ^{Basal Ganglia}
- **A**sterixis.
- **D**isturbed personality & behaviour: childish attitude & irritability.
- **D**isorientation for: time, place, persons.

←
بیماری
روان
و تفکر

2. Coma:

- Irritable coma with features of liver failure & finally death.

→ also due to sympathetic ↑ (hypoglycemia) 3/15 →

↓
لوهای
بسیار
جدا

- Classifications & Grading:

- Recently, hepatic encephalopathy has been classified according to severity into:

"West Haven Criteria"

GRADE	MENTAL STATUS	NEUROLOGICAL FINDINGS
Grade 1	Mild lack of awareness Inverted sleep rhythm	Astrexia may be detected
Grade 2	Lethargy or apathy Inappropriate behavior	Astrexia obvious = drowsy + ↓ cognitive function
Grade 3	Somnolence Marked disorientation	Astrexia obvious
Grade 4	COMA	deep COMA 3/15

#

- Also, in the World Congress of Gastroenterology 1998 in Vienna, hepatic encephalopathy was subdivided into 3 subclasses, Type A, B and C:

مش
صحت
تدریجی
astrexia

acc
to
etiology

- Type **A** (= **A**cute): associated with **A**cute liver failure.
- Type **B** (= **B**ypass): is caused by port – systemic shunting (**B**ypass) without associated intrinsic liver disease.
- Type **C** (= **C**irrhosis): occurs in patients with **C**irrhosis.

lcf

و ده می
Bilth.
(فایل مناسبت)
Pentel
HTN
White Knight Love

HEMATOLOGICAL MANIFESTATIONS

A. Anemia:

1. Microcytic hypochromic anemia: due to repeated hemorrhages, e.g. oesophageal varices.
2. Normocytic normochromic anemia: due to acute hemorrhage or hypersplenism.
3. Macrocytic anemia: due to deficiency of folic acid or vitamin B12.

B. Bleeding tendency:

I. Defect in the clotting factors

- PROTHROMBIN II
- Fibrinogen. I
- Factors V, VII, IX, X,

"Deficiency",

especially:

not due to ACD
لأنه من
inflamm
malignant

اعطاء vit K لا يفيد
لأنه ليس من نقص
الخلايا القليلة
الكل. ادى بلازما من
في 13

II. Defect in the platelets:

1. Thrombocytopenia: "Decreased platelets": due to hypersplenism.
2. Thrombocytopathy: "Diseased platelets". due to coating by toxins

vit K dependant

C. Cytopenia:

Pancytopenia due to hypersplenism.

ENDOCRINAL MANIFESTATIONS

1. In males: [Feminization]

- Gynecomastia.
- Testicular atrophy, impotence, ↓ libido, sterility.
- Feminine distribution of suprapubic hair. ↓ hair between umbilicus & suprapubic

2. In females: [Defeminization]

- Atrophy of the breasts.
- Amenorrhea, ↓ libido, sterility.

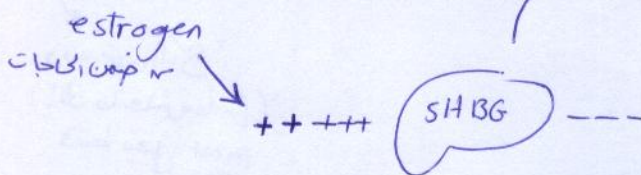
اول حاجه تقول

- The mechanism of these endocrinal manifestations is unclear, but may be due to:

- Increased estrogen. decreased its destruction
- Decreased testosterone.
- Increased sex hormone-binding globulin (SHBG).

♀ binds to estradiol in active form & blocks its action on receptors

♂ binds to testosterone & blocks its action on receptors
testosterone ~ White Knight Love



DISTURBED CARBOHYDRATE METABOLISM

- In chronic liver failure: IGT & rarely secondary diabetes.
 Impaired Glucose Tolerance
 mild Hyperglycemia
 Hyperglycemia
- In acute liver failure: Hypoglycemia.

due to insulin resistance due to ↓ Receptors
 Fat muscles liver (destroyed)

↓ glycogen

Sudden failure of glycogenolysis

INCREASED INCIDENCE OF INFECTIONS

Spontaneous Bacterial Peritonitis " SBP "

Definition

- Infected ascitic fluid with no clear cause for peritonitis.
- It is probably due to ↓ antibacterial activity of the ascitic fluid in cirrhosis.

Clinical picture

- It occurs in about 10 % of cirrhotic patients with ascites.
- Sudden deterioration of a cirrhotic patient & encephalopathy with no obvious ppt. factor.
- **Fever, abdominal pain & tenderness.** + refractory ascites
 40°C

Ascitic fluid analysis

- Polymorph count usually exceeds: 250 cells / cmm.
- Ascitic fluid culture is usually: positive for organisms.
- The infecting organisms are usually: gram negative organisms.
 may be streptococci

Treatment

- Broad spectrum antibiotics: Cephalosporins, Ciprofloxacin.

INVESTIGATIONS

1. Liver function tests:

- Albumin
 Prothrombi
 ↓
- Plasma proteins: ↓ albumin, ↑ globulin, reversed A/G ratio.
- Prothrombin time: Prolonged, & not corrected by parenteral vitamin K.
- Serum bilirubin: ↑ both direct & indirect.
 & INR ↑
 due to ↓ clotting factors
- Serum enzymes: ↑ transaminases & ↑ alkaline phosphatase.
 AST ALT
 is age ↑
 Lcf ↑

severe ↑

↑ due to destruction of cells (slowly gradually) White Knight Love

2. Investigations for the cause:

- e.g. Abdominal ultrasonography or CT scan for cirrhosis.
e.g. HEPATITIS MARKERS for hepatitis.

3. Investigations for the precipitating factors:

- e.g. serum potassium.

4. Investigations to exclude other causes of coma:

- in case of hepatic encephalopathy, e.g. brain imaging & lab tests.

TREATMENT

1. TTT of the cause:

stop drug: eg: INH if possible.

2. TTT of the precipitating factors:

if present. eg: GIT Bleeding

3. TTT of ascites:

if liver failure is minimal & no encephalopathy.

4. TTT of bleeding tendency:

FFP to replace coagulation factors.
fresh frozen plasma

5. TTT of hepatic encephalopathy:

Care of the comatosed

A) Diet:

- Protein restriction: 1 gm / Kg / day. (60 gm في اليوم)
- Carbohydrate excess: to decrease protein catabolism.
- Potassium excess: fruit juices. (except apple juice)

B) Bowel sterilization:

- Neomycin: 1 gm / 6 hours to prevent action of intestinal bacteria on proteins.
- Metronidazole: 0.5 gm / 6 hours in cases of associated renal impairment.

C) Colonic lavage:

- Enemas to wash the intestinal contents & thus decrease the production of toxic substances by the effect of bacteria on proteins.

D) Lactulose:

- It is a synthetic disaccharide metabolized by the intestinal bacteria causing:
 - Production of acids, which will ↓ the fecal pH & change the bacterial flora.
 - Laxative effect washing the intestinal contents.

E) Other measures:

- L – ornithine L – aspartate (LoLa): **“Hepamerz”**

- It reduces the level of ammonia.

- Drugs of doubtful value (Bad drugs): **“3 B”**

- **B**ranch ed aa: to correct their level.
- **B**romocryptine & L-dopa: to improve neurotransmission.
- **B**enzodiazepine antogints.

- Sedatives:

- They should be avoided, but if necessary, give very small doses of: diazepam.
- Morphine is absolutely contraindicated.

- Artificial hepatic support: **“charcoal hemodialysis”**

- Charcoal filters are used to adsorb toxins from the blood of the patient.

6. HEPATIC TRANSPLANTATION:

- It is the only curative FINAL treatment for liver failure.

also
Parkinsonism
Acromegaly
LCF (encephalopathy)

ارابط جليل
وايه نرا

agressive جداً
ليك
Hepatitis
فيك ويك
تسك سة البيرة وهو عيان

ACUTE (FULMINANT) HEPATIC FAILURE

DEFINITION

لازمی سوالات از خری سکر SQ

- Severe hepatic failure with ENCEPHALOPATHY developing in less than 8 weeks in a patient WITH NO PREVIOUS LIVER DISEASE.

ETIOLOGY

کانونی جبهه
و جبهه سمت راست
Liver
نمایان

- Severe, Sudden, Massive necrosis of the liver cells due to:

1. Infections:

If Pregnant

chronic disease

- Viral hepatitis: E, B, C, D.
- Other viruses causing hepatitis: EBV, CMV, HSV, Yellow fever.

2. Drugs & toxins:

- Alcohol.
- Halothane.
- INH & paracetamol.
- Carbon tetrachloride, DDT, phosphorus.

close dependant causes acute not chronic

3. Acute fatty liver of pregnancy.

massive fatty degeneration pregnant

4. Acute Budd – Chiari.

5. Reye's syndrome: hepatic injury, CNS injury, hypoglycemia: in children.

viral infection aspirin sensitivity

CLINICAL PICTURE

آیا
chronic
or
acute
؟

- Hepatic encephalopathy: severe & early & ends in coma.
- Fetor hepaticus.
- Jaundice: mild in early stages & becomes deep in late stages.
- Fatal course: the condition usually ends by death due to:

- Severe encephalopathy.
- Severe bleeding.
- Severe septicemia.
- Severe hypoglycemia.
- Severe multiple organ failure: circulatory, respiratory, renal.

bypass
failed detoxification

Hypoglycemia (failed glycogenolysis)



ARF
due to ARDS
due to sepsis

White Knight Love

HCO → Hypoalbuminemia is a marker of chronic Lf liver disease 42
 → not a marker of Acute liver disease

INVESTIGATIONS

1. Liver function tests:

زی
هنا

- Serum bilirubin: is elevated & is used to assess the prognosis. ←
- Serum albumin: is not affected. ←
- Serum transaminases: ↑ early, but may ↓ later due to massive necrosis. ←
- Prothrombin time: is prolonged & is used to assess the prognosis. ←

as
life
albumin
↓

یمن یقصر
فترة طویل
في الدم
فمن هیما
نقص
albumin
فمن مایوت

2. MRI & CT of the brain:

to exclude brain lesions. (Coma Central) Causes

3. Investigations for complications:

e.g.

- Blood glucose, creatinine, Na, K, blood gases.

ما هو عبارة عنه
Hepatic Enc.

4. Investigations for the cause.

eg viral markers

TREATMENT

1. Hospitalization in the ICU:

and care for the comatosed.

2. Treatment of the cause:

if possible. eg: correction of ↓ G

3. Treatment of hepatic encephalopathy.

استرج مای

4. Treatment of complications.

5. Artificial hepatic support:

“ charcoal hemodialysis ”

- Charcoal filters are used to adsorb toxins from the blood of the patient.

6. Hepatic transplantation:

- It is the only curative treatment for liver failure.

استرج مای hemiplegia

ASCITES

= Peritoneal effusion

DEFINITION

- Excessive accumulation of fluid in the peritoneal cavity.

ETIOLOGY

I. TRANSUDATIVE ASCITES

non inflamm.
"Accumulation of transudate"

1. Organ failure:

The most common causes

- Liver cell failure:
- Right sided heart failure.

The most common cause

ascites before edema
edema before ascites

2. Portal hypertension:

- a) Suprahepatic causes: RVF, Pericardial eff, High IVC obstr. & Budd-Chiari.
- b) Intrahepatic causes: Liver cirrhosis, VOD, Bilharzial periportal fibrosis.
- c) Infrahepatic causes: Portal vein obstr, e.g. thrombosis or malignancy.

3. Hypoalbuminemia:

- Nephrotic syndrome.
- Nephritic syndrome.

proteinuria
nephrotic → gradual onset Ascites
nephritic → Acute onset Ascites

- Nutritional: severe malnutrition & severe malabsorption.

4. Miscellaneous:

- Meig's syndrome. → Rt pleural effusion, ovarian tumor, Ascites
- Myxoedema. → caps permeability due to Mps damaging caps wall

inflammatory

II. EXUDATIVE ASCITES

"Accumulation of exudate"

1. Peritoneal diseases:

- Infections: TB peritonitis, SBP.
- Malignancy: Malignant deposits in the peritoneum.

indications: w/pt of cortisol ttt & anti TB

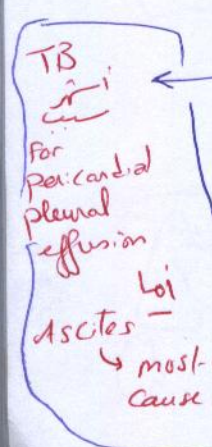
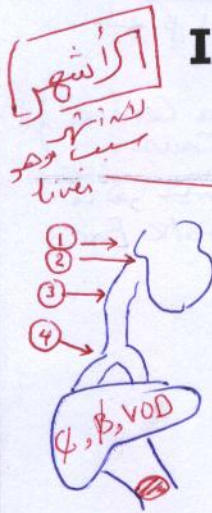
Hgic malignant cells
Massive rapid accumulation after aspirate

2. Pancreatic ascites:

which is a complication of pancreatitis.

pancreatitis → damage of pancreatic duct
Rupture of pancreatic pseudo cyst
or
Formation of pancreatic fistula
Fistula between pancreas & peritoneum
Leak of juices into peritoneal cavity

White Knight Love



III. CHYLOUS ASCITES

- Obstruction of the thoracic duct:
- Rupture of the thoracic duct:

"Accumulation of lymph"

tumours or filariasis.
trauma.

IV. HEMORRHAGIC ASCITES

- Hemorrhagic blood diseases.
- Trauma. (Bleeding from other orifices)

"Accumulation of blood"

- TB
- Malignancy

CLINICAL PICTURE

ascites
ery effect
cause

1. CLINICAL PICTURE OF ASCITES

Symptoms

"especially in tense ascites"

- Abdominal distension.
- Abdominal discomfort. may be pain due to stretch of peritoneum
- Dyspepsia. = discomfort after meals (as Cirrhosis $\rightarrow$ ↓ gastric destruction
Congestive gastropathy)
- Dyspnea. ↓ Compliance of lungs, pleura & chest wall & may be basal lung collapse
- Development of abdominal hernia. (chronic ↑ IAP)

↓ dull
↓ air entry

Signs

حوار Clinical

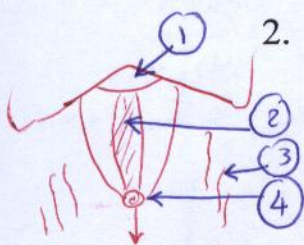
Inspection

1. Abdominal distension (mainly in the flanks) ± visible veins.

due to stretch of abd. wall
دول غير
- Dil v.v.
الى!
- Palpable
- elevated
- tortuous

2. Signs of chronic increase in the intra-abdominal pressure:

- Subcostal angle: widening.
- Umbilicus: everted, shifted downwards, ± umbilical hernia.
- Striae: stretch marks.
- Divarication of the recti.



3. Dilated veins on the abdominal wall:

- Portal hypertension (Caput Medusae).
- IVC obstruction (Cause of ascites or Effect of ascites).

* protuberant abdomen
* thin extremities (wasting)
spider man

Palpation

1. Transmitted fluid thrill:
2. Dipping method to feel liver & spleen:
3. Abdominal masses may be felt:

in tense ascites.

in tense ascites.

in TB & Malignancy.

to displace fluid

ملاحظة
فقط تقول
حالة

① TB lymphadenitis
② Adhesions & fibrosis of peritoneum (short White Knight Love)

IVC

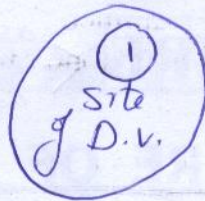


Caput medusa



التفرع

at periphery



around Umbilicus

②

milking

below

Umbilicus

Above downwards

below upwards

لأنه فوق

كل واحد من يتفرع

Percussion

1. Shifting dullness: is the most important clinical sign of ascites:

Value detects moderate amount of ascites (500 ml).

Test

First step

1. The patient is examined in the supine position.
2. Direct percussion is done over the abdomen, from the umbilicus to the flanks.
3. Locate the border between resonance & dullness.
4. **Positive test**: Percussion note is resonant over the umbilicus and dull over the flanks.

if Resonant
all over

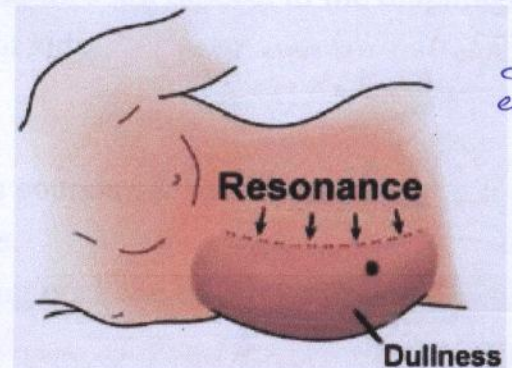
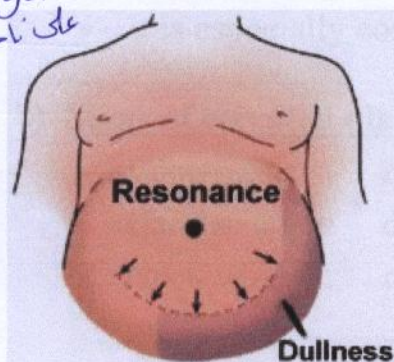
Note: The resonance over the umbilicus occurs in ascites because bowel floats to the top of the abdominal fluid at the level of the fluid surface.

Second step

5. The patient then is rolled on his side away from the examiner, and percussion from the umbilicus to flank area is repeated.
6. **Positive test**: When ascites is present, the area of dullness will shift to the dependent site. The area of resonance will shift toward the top.

Note: The test should be repeated on the other side.

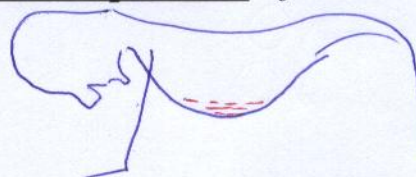
على سطح السائل
encysted
على ناحية واحدة



don't
exclude
p/s
for

2. Percussion in knee-elbow position: for minimal ascites (120 ml).

if ve
↓
u/s



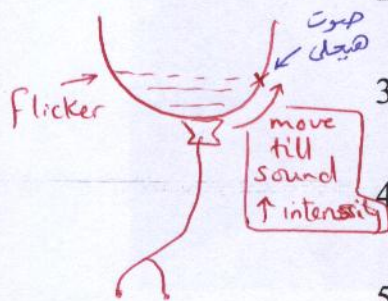
WhiteKnightLove

Auscultation

1. Puddle sign: “Auscultating percussion”

Value detects minimal amount of ascites (120 ml).

Test



1. The patient lies in the knee – elbow position.
2. Place the stethoscope on the most dependent part of the abdomen.
3. The nearby flank is percussed repeatedly by light flicking of constant intensity.
4. Gradually move the stethoscope towards the flank opposite the percussion.
5. An increase in the intensity of the sound heard by the stethoscope indicates that there is a fluid level.

2. Venous hum: “heard in portal hypertension”

- It is a soft systolic murmur over dilated para-umbilical vein.
- It is heard between the umbilicus & the xiphi-sternum.

2. SECONDARY EFFECTS OF ASCITES

a) Pleural effusion:

- It is probably due to passage of fluid through defects in the diaphragm.
- It is especially common on the right side. → (small anatomical defects on Rt side)

b) Signs of elevated diaphragm:

- Lung bases: diminished breath sounds & dullness.
- Cardiac apex: displaced upwards.
- Neck veins: congested pulsating neck veins.

سپر حیدر
back

→ as elevated diaphragm → ↑ intrathoracic pressure → ↓ VR (Congestion)

3. FEATURES OF THE CAUSE OF ASCITES

e.g. Features of liver cell failure & portal hypertension in case of liver cirrhosis.

کتاب
نویس

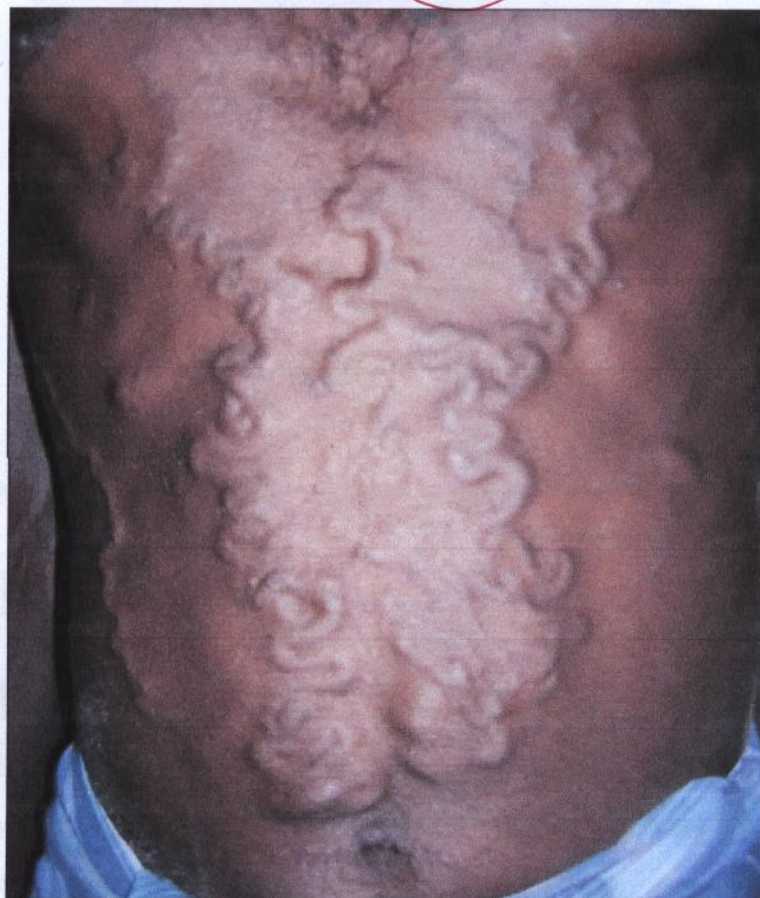
Jaundice
- fetor
- gynecomastia

varices

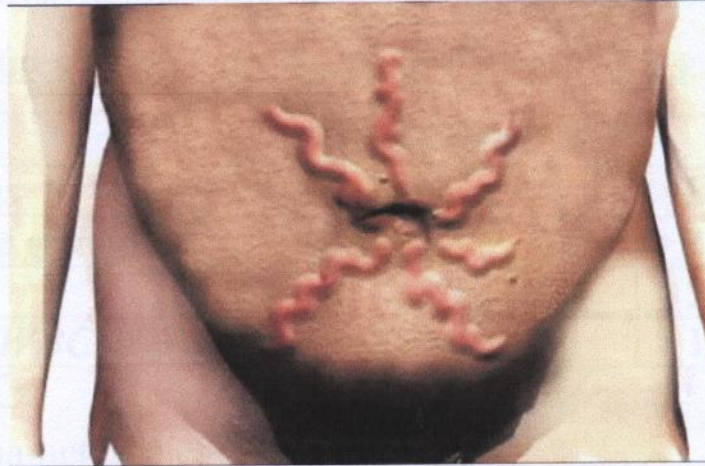
Tense ascites with visible veins



Ascites with dilated veins



Ascites with caput medusae



Ascites with umbilical hernia



INVESTIGATIONS

انہی کے لئے
نوعی
سیر

I. Investigations to detect the presence of ascites:

- Abdominal ultrasonography & Abdominal CT:

- Detect the presence of even minimal ascites. (good +ve & good -ve)
- Differentiate free ascites from encysted ascites.
- Detect the cause, **e.g.** liver cirrhosis.

II. Investigations to detect the type of ascites:

- Aspiration of ascitic fluid:

- Analysis for: *physical, chemical, cytological, bacteriological characters:*

a) Features of exudate & transudate effusions:

	TRANSUDATE	EXUDATE
1 Proteins	< 3 gm %	> 3 gm %
2 Specific gravity	< 1016	> 1016
3 LDH	< 200 IU / L	> 200 IU / L
4 Cells (WBCs)	< 1000 / cmm	> 1000 / cmm

1- age
V-recent
5 → SAAG

Serum
Ascitic
Albumin
Gradient

فقد مع جـ د
ascites
مع جـ د
pleural or pericardial

NB Characteristics of TB effusion:

1. Exudate: rich in Lymphocytes & RBCs.
2. TB bacilli can be detected by:
 - *Staining:* ZN stain.
 - *Culture:* Lowenstein-Jensen medium or BACTEC.
 - *Animal Innoculation:* Guinea pig.

b) Features of Spontaneous Bacterial Peritonitis:

- The ascitic polymorph count exceeds 250 cells / cmm.
- The ascitic fluid culture is positive for organisms.
- The infecting organisms are usually: gram negative organisms.

c) Features of Hemorrhagic ascites:

- The fluid is bloody & contains many RBCs.

d) Features of Chylous ascites:

- The fluid is milky white & contains many fat, clears on addition of ether & stains orange with: Sudan III.

e) Features of malignant ascites: see later.

III. Investigations to detect the cause of ascites:

- For liver cirrhosis: *eg* liver function tests.
- For heart failure: *eg* echocardiography.
- For TB & malignancy: *eg* laparoscopy & biopsy.

"Recent parameter in the diagnosis of ascites"

Serum – Ascitic Albumin Gradient (SAAG)

- Definition:

The serum-ascitic albumin gradient (SAAG) is calculated by subtracting the albumin concentration of the ascitic fluid from the albumin concentration of a serum specimen obtained on the same day.

- Clinical value:

SAAG is directly related to the portal pressure:

	Transudate	Exudate
Serum Albumin	5	4
Ascitic Albumin	2	3.5
gradient	3	0.5

1. Ascitic patients with SAAG ≥ 1.1 g/dl have portal hypertension (transudative ascites).

2. Ascitic patients with SAAG < 1.1 g/dl have non-portal hypertension etiology (exudative ascites, mostly)

DIFFERENTIAL DIAGNOSIS

A. From other causes of abdominal distension:

5 F

- **Fat:** (Obesity): No shifting dullness.
- **Flatulence:** Hyper-resonance all over the abdomen.
- **Foetus:** (Pregnancy): Signs of pregnancy are present.
- **Full urinary bladder:** Suprapubic tenderness + catheter will release the urine.
- **Fluid in a cyst (Ovarian cyst):**
 - Distension is mainly central.
 - Umbilicus is shifted upwards.
 - Central dullness & resonant flanks.
 - No shifting dullness. *as not free fluid*

B. DD of the cause of ascites.

اشرح + most imp criteria of each cause

TREATMENT

eg: Anti TB for TB

I. Treatment of the cause of ascites.

II. Treatment of ascites in cases of liver cirrhosis:

General Rule

- Always keep the patient **Wet & Wise** and not **Dry & Drowsy**.
- Treat ascites only if: mild liver failure & no encephalopathy.

A) GENERAL MEASURES: (Conservative)

1. Bed rest: better renal perfusion in recumbancy will lead to diuresis.

2. Diet:

- Salt: restriction. → if severe restriction → dilutional ↓ Na [low salt syndrome]
- Fluid: restriction in severe cases of hyponatremia.
- Protein: high protein diet (protein is restricted if there is encephalopathy).

3. Follow up:

- Daily measurement of: urine volume & body weight.
- Daily measurement of: electrolytes (Na & K) & renal functions.

B) MEASURES TO REMOVE THE ASCITIC FLUID:

4. Diuretics:

- Indication: If weight loss is less than 1 Kg after 4 days on diet control.

• Drugs:

a) At first: Potassium – sparing diuretics, e.g. aldactone (100 – 400 mg/day).

Add b) If there is no improvement: Lasix (40 – 120 mg / day) + K supplement.

c) In resistant cases: IV Mannitol or IV infusion of Dopamine.

5. Albumin (IV):

may be given to correct hypoalbuminemia.

6. Tapping of ascites:

- Indication: [Tense ascites]

- Respiratory distress.
- Impending rupture of umbilical hernia.

- Contra-indication: Severe liver failure, encephalopathy, renal failure.

- Volume: 4 – 5 litres at one time combined with IV albumin.

لا زود الحياءه قاعه
وخط الابره
in dependant part

داخل على
encephalopathy

White Knight Love
↓↓↓ Blood flow to Kid

↑↑↑
↑↑ Renal
Blood
flow

Hepatorenal
syndrome

لا زود الحياءه

بسط كود

استطارة

داخل على

بسريرج
او هيفرق

ORAI advantages
of PSD →

H-E. → ↓K حافض

برغم انه حافض
للكتروليتات
فلا يفضل
في حالات
الاصابة
بالاصابة
باصابة

1. Aldosterone
antagonist

resistant to ttt

C) TREATMENT OF REFRACTORY ASCITES:

- Definition:

- Ascites unresponsive to:
full dose 400 mg of spironolactone *plus* *full dose* 120 mg of frusemide daily for two weeks.

- Resistance to treatment may be due to:

- ملح زايد* Lack of salt restriction: treated by adequate salt restriction.
- لازم تصلي* Severe hypoalbuminemia: treated by IV albumin. *as only removes water (not salt)*
- ملح ناقص* Dilutional hyponatremia: treated by fluid restriction & IV mannitol.
- Serious problems as SBP, TB peritonitis or malignant ascites: ttt the cause

- Severe terminal cases may be treated by:

- Le Veen shunt: *= Caval* (peritoneo – venous shunt)
 - Technique:
 - A catheter with one way valve is placed between the peritoneal cavity and the SVC to drain the ascitic fluid into the circulation.
 - Complications:
 - Hypervolemia, pulmonary edema, infection, DIC. *septicemia for abn. activation of clotting factors by F.B.*
- Transjugular intrahepatic portosystemic shunt: (TIPS)
 - Technique:
 - It is an artificial channel in the liver from the portal vein to a hepatic vein to reduce the portal pressure (shunting).
 The catheter is introduced percutaneously via the IJV.
 The shunt is maintained open by a metal stent.
 - Complications:
 - Hepatic encephalopathy, TIPS stenosis. *Reaction fibrosis around*
- Ascites ultrafiltration & reinfusion:
 - Ultrafiltration: removes the ascitic fluid & concentrates it.
 - Reinfusion: returns the fluid to the patient IV.
- Hepatic transplantation.

*↓ portal pressure
 ↓ toxins by pass*

TUBERCULOUS PERITONITIS

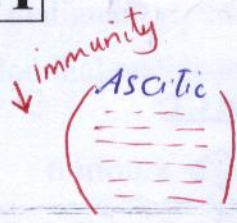
not most common cause of ascites
من يتلف في البال

ETIOLOGY

- Direct spread from: TB mesenteric LN, TB enteritis, TB salpingitis.

PATHOLOGY

- TUBERCLES:

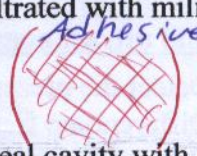
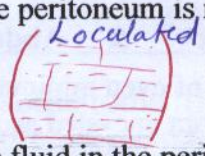


- TYPES:

according to immunity

1. Ascitic type:
2. Loculated (encysted) type:
3. Adhesive type:

The peritoneum is infiltrated with miliary tubercles.



Granuloma

free fluid in the peritoneal cavity with NO ADHESIONS.

ADHESIONS divide the peritoneal cavity into many loculi: each containing ascitic fluid.

MARKED ADHESIONS with no fluid formation.

- TABES MESENTERICA:

TB infection of the mesenteric LN may occur.

- THE OMENTUM:

may form a rolled (sausage - like) mass due to adhesions.

COMPLICATIONS

1. Intestinal obstruction: due to ADHESIONS.
2. Intestinal fistula.
3. Tense asites.
4. General complications of TB (refer to ahest).

استشر

CLINICAL PICTURE

Symptoms

General:

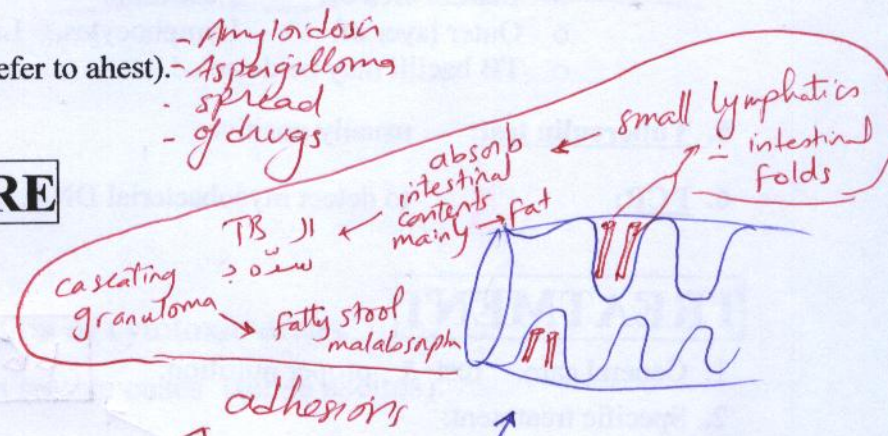
- Symptoms of TB toxemia.

Abdominal:

القلادة
يمكن

- Constipation:
- Diarrhea may occur due to:
- Abdominal discomfort or pain.
- Abdominal distension.

استشر للقاعدة
ORAL

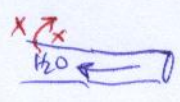


is common.

TB enteritis, obstruction of the lacteals, Addison's disease.

infective diarrhea

reaches adrenal
Addison's → chronic diarrhea
↓↓↓ cortisol
White Knight Love



Signs

General:

- Fever, weight loss, weakness, pallor, toxic facies.

Abdominal:

- Ascites. *Caseation*
- Doughy sensation, tenderness, mild rigidity.
- Palpable masses: rolled omentum or tuberculous LN.

INVESTIGATIONS

1. Abdominal ultrasonography & Abdominal CT:

- Detect the presence of even minimal ascites.
- Differentiate free from encysted ascites.
- Detect LN enlargement.

2. Abdominal Plain X-ray:

- Calcified mesenteric LN.
- Multiple fluid levels in the loculated type.

3. Aspiration of ascitic fluid:

- Straw – coloured & may be turbid.
- Features of an exudate + increased lymphocytes + increased RBCs.
- The fluid should be subjected to:
 - Direct smear stained with ZN stain.
 - C & S test with Lowenstein medium.
 - Animal inoculation in guinea pig.

4. Laparoscopy or Laparotomy:

- They will show peritoneal tubercles which should be biopsied.
- The biopsy will show the characteristic granulomatous caseous lesion of TB:
 - Central area of: Caseation.
 - Outer layer of: Lymphocytes, Langhan's giant cells, Fibroblasts.
 - TB bacilli may be detected.

5. Tuberculin test: usually positive.

6. PCR: to detect mycobacterial DNA.

from ascitic fluid

TREATMENT

1. General care: rest & proper nutrition.

2. Specific treatment:

- Anti-tuberculous drugs.
- Corticosteroids: prednisone 1 mg / Kg / day for 1 month: "to reduce inflammation & reduce fibrosis".

3. Treatment of complications: e.g. intestinal obstruction, tense ascites.

MALIGNANT ASCITES

ETIOLOGY

1. Secondary:

- To abdominal malignancy: due to direct infiltration or shedding of malignant cells.
- To extra – abdominal malignancy: due to lymphatic or blood spread.

2. Primary: tumours e.g. mesothelioma (rare).

CLINICAL PICTURE

1. Manifestations of the PRIMARY TUMOUR.
2. Features of malignant ascites: massive, rapidly re-accumulating after tapping.
3. Abdominal masses may be detected & nodules around the umbilicus may be present.

Sister Mary Joseph

ASPIRATION OF ASCITIC FLUID

- Features of exudate: massive, hemorrhagic, may contain malignant cells.

TREATMENT

- Palliative:

- Intraperitoneal injection of cytotoxic drugs.
- Tapping of ascites in severe cases (tense ascites).

ORAL : ? mixed أسباب

Clinical

(+)

نخري

56

- eg ① Biliary cirrhosis Started obstructive jaundice
ended by Cirrhosis (hepatocellular) jaundice
eg ② Hemolytic anemia Started by Hemolytic jaundice
ended by
eg ③ Viral hepatitis Started hepatocellular
ended by obstructive (edema of liver cells obstruct ducts) jaundice

JAUNDICE

discuss
أي واحد من الأنواع

N: 1 mg/dl (total)

1 DEFINITION

- Yellow discolouration of the: sclera, skin & mucous membranes due to increased serum bilirubin.
- Jaundice is clinically detectable when: serum bilirubin exceeds 2.5 mg / dl.

2 TYPES

1. Hemolytic jaundice.
2. Obstructive jaundice.
3. Hepatocellular jaundice.
4. Mixed jaundice.

If 1.5 or 2 mg/dl
not normal not jaundice
called: lab. hyperbilirubinemia
subclinical jaundice

في ضوء النور
وان كان كلام نارغ

oral

في غيابه أصفر ناتج من jaundice ← أو
Carotenemia
no discoloration eyes urine stool
myxedema

also
Cyanosis
not > 5g reduced
Hb
not cyanosed
but hypoxic

3 INTRODUCTION

In the Blood: Bilirubin formed as a result of RBCs destruction will bind to albumin forming:

normally
never traces of Bilirubin
in urine

1. **HAEMBILIRUBIN: "Unconjugated OR Indirect"**
- It cannot be excreted through the kidney (big molecular weight & water insoluble).

In the Liver: Liver cells will deal with this bilirubin through 3 steps:

- **Uptake.**
- **Conjugation:** of bilirubin to glucuronic acid by glucuronyl transferase enzyme to form:
2. **CHOLEBILIRUBIN "Conjugated OR Direct"**
Can be excreted by kid. Small MW & water soluble
- **Excretion:** of cholebilirubin into the bile canaliculi to reach the intestine.

3. **In the intestine:** cholebilirubin is reduced to stercobilinogen which passes in 3 routes:
 - **Entero-hepatic circulation:** part is absorbed to reach liver & is then re-excreted to intestine.
 - **Systemic circulation:** part passes to systemic circ. & is excreted by kidneys as urobilinogen.
 - **Stools:** part is oxidized into stercobilin & passes in stools giving it the brown colour.

4 PATHOGENESIS

I. HEMOLYTIC JAUNDICE:

- Increased hemolysis of RBCs → increased haembilirubin.
- The liver cannot uptake all the increased haembilirubin completely, therefore: part of haembilirubin is retained in the blood → increased serum bilirubin → **Jaundice** (mild)
- Bilirubin is not present in urine: **"Acholuric jaundice"** (no urobilinogen in urine)
- Excessive haembilirubin will be converted to cholebilirubin & excreted by the liver to the intestine → increased stercobilinogen → **Dark stools** (dark stools)
- Increased stercobilinogen absorbed from intestine → increased urobilinogen, therefore: **Urine is normal in colour but darkens on standing.**

بقدر مكانه
بس مش أخضر
أوى لانه
أخف ملونه
chole

قلو الاستاج زاد أكثر (hemolytic)
هيفر لونه stool أغمر
هو من stercobilinogen بس لما راحت كبد اتغير اسم
أما التركيب هو هو

مش هو
ده إلى
بيصفر
البول
أخضر
أخضر
السبب
Urochrome
Pigment

Hepatocellular

normal
الأنس هتتلف هو كفاية
انغم دك

لو لقيت عند غيابه
dark stools
لحق أكيد
stercobilinogen
urobilinogen
jaundice
White Knight Love

Q: How to answer any type of Jaundice?
general frame

- ① Etiology
 - ② Pathogenesis
 - ③ CLP
 - ④ Investigations
- eye stool urine general cause
blood stool urine general cause

57

II. OBSTRUCTIVE JAUNDICE:

- Biliary obstruction will prevent cholebilirubin to reach the intestine, therefore: cholebilirubin regurgitates into the blood → increased serum bilirubin → **Jaundice.**
- Excessive cholebilirubin appears in urine → **Dark urine.**
- Bile salts regurgitate into the blood & appear in urine.
- Bile does not reach the intestines → **Pale stools** (clay-coloured, bulky, offensive, greasy).
- There is decreased stercobilinogen, decreased urobilinogen.

fat absorption

III. HEPATOCELLULAR JAUNDICE:

- Liver cells are not able to: uptake, conjugate & excrete all the bile pigments.
- A part of haembilirubin is retained in the blood & a part of cholebilirubin regurgitates into the blood → increased serum bilirubin (direct & indirect) → **Jaundice.**
- Cholebilirubin appears in urine → **Dark urine.**
- Stercobilinogen is decreased → **Pale stools.** (↓ excretion)
- Urobilinogen is increased because the diseased liver cannot fully re-excrete the absorbed stercobilinogen.

Urobilinogen not ↑ due to systemic circ. absorption but ↑ due to stercobilinogen enterohepatic circ. to liver and liver can't excrete it (↑ in blood) → to kidney as urobilinogen

HEMOLYTIC JAUNDICE

الذكر بالقرص

1 ETIOLOGY

- Causes of hemolytic anemia (refer to hematology).

2 pathogenesis

3 CLINICAL PICTURE

1. Jaundice: is usually mild (lemon yellow).
2. Stools: dark.
3. Urine: normal in colour but darkens on standing.

4. Features of hemolytic anemia: e.g. HSM, gall stones, leg ulcers, hemolytic crises.

5. Features of the cause, e.g. thalassemia.
PNH
SCA

CLP	lab
↑ RBCs destroy	
↑ hemobilirubin	mild jaundice ↑ indirect
not in urine as big insoluble	no Bilirubin
cholebilirubin	↑ stercobilinogen
stercobilinogen	dark stool
urobilinogen	normal urine ↑ uro

4 INVESTIGATIONS

1. Serum bilirubin: ↑ increased mainly the indirect (hemo)
2. Stools: increased stercobilinogen.
3. Urine: increased urobilinogen, absent bilirubin.

4. Blood picture of hemolytic anemia.
5. Investigations for the cause, e.g. Hb electrophoresis for thalassemia.

Hb electrophoresis (Hb S) SCA
Flow cytometry PNH

except in thalassemia micro, hypo

Oral H.A. → intra hepatic → small pigment granules
 extra hepatic → stone CSD

OBSTRUCTIVE JAUNDICE

(CHOLESTATIC JAUNDICE)

Bile stagnant

اكثر جراحه
 لکن جاء

Oral 1000
 Causes of intermittent jaundice

1 ETIOLOGY

surgical

I. EXTRAHEPATIC OBSTRUCTION:

- In the lumen:**
 - Stones in the common bile duct or common hepatic duct.
 - Ascaris worms: very rare.
- In the wall:**
 - Strictures: e.g. post-operative. or postinflammatory (pancreatitis, sclerosing cholangitis)
 - Tumours: e.g. cholangiocarcinoma. (due to central necrosis)
- Pressure from outside:**
 - Cancer head of pancreas.
 - Cancer ampulla of Vater.
 - Enlarged LN in porta hepatis.

malignant

intermittent jaundice

red

medical

II. INTRAHEPATIC OBSTRUCTION:

- Primary biliary cirrhosis.
- Pregnancy jaundice. V.V.V. rare
- Cholestatic type of viral hepatitis.
- Chronic hemolytic anemia. → small pigment granules
- Cancer liver: HCC or secondaries.
- Chlorpromazine & (CCPs.)
- Helminthes: Fasciola.
- Hereditary disorders: (stasis without true obstruction)
 - Dubin Johnson syndrome: Hereditary defect in bile excretion with greenish black liver.
 - Rotor syndrome: Hereditary defect in bile excretion with normal colour of the liver.

in 3rd trimester not mechanical obstruction but due to prostaglandin in some susceptible
 stagnation ↓ flow of bile as hepatic
 major transport of antineoplastic

RBCs	CIP	lab
hemobilirubin ↓ ↑ cholebilirubin ↓ In urine as small soluble obstruction	deep jaundice dark urine	↑ direct Yes Bilirubin
↓ stercobilinogen ↓ ↓ urobilinogen	pale stool dark urine ↑ cholebilirubin	↓ stercob ↓ uro

على سطح الكبد
 الحصى الصفراء
 لو قوت
 يقول
 hepatocellular to cholestatic
 as it is
 = swollen cells
 ↓ function of cells (hepatocytes)

Pathogenesis

CLINICAL PICTURE

- Jaundice: is usually deep (olive green).
- Stools: pale (clay-coloured, bulky, offensive, greasy).
 bulky: fat
 greasy: fat
 offensive: bacteria on fat
 steatorrhea (Fatty diarrhea)
- Urine: dark (frothy).
 due to Bile salts ↑ in blood (Physical characteristics)
 Cholebilirubin
- Features of obstructive jaundice: Pruritus, Bradycardia, Bleeding tendency, Bone affection.
- Features of the cause, e.g. gallstones.
 - Extra intra I PBC
 - FLQ
 - FLQ
 - direct inhibition of SAN by bile salts
 - ↓ int K & connected by injection
 - ↓ int K
 - Xanthomata
 - nodules or plaques, yellowish, on inner canthus eye rich in lipid especially cholesterol
 - ↑ cholesterol (formed & metabolized by liver and excreted by liver into intestine) → Oral
 - endogenous → in liver
 - exogenous → in intestine
 - White Knight (blue)

Xanthomata

دوبين و جونسون
 deposition of melanin like pigment - liver

2
3

4 INVESTIGATIONS

1. Serum bilirubin: increased mainly the direct.
2. Stools: decreased stercobilinogen.
3. Urine: decreased urobilinogen, bilirubin is present. *chole N: no bilirubin*

4. Investigations suggestive of obstructive jaundice:

- فح* Liver functions: elevated enzymes (ALP, GGT, 5 - nucleotidase), prolonged PT that is corrected by parental vitamin K, increased cholesterol with normal esterification. *not connected if hepatocellular*
- انكبات* Ultrasonography & CT: dilated intrahepatic biliary radicles in extrahepatic obstruction. reveals the cause of extrahepatic obstruction. *cancer head porta hepatis ampulla*
5. Investigations for the cause, e.g. MRCP, ERCP, PTC for: detection of the site of obstruction. Anti-mitochondrial antibodies for: PBC. *normal esterification*
- intra* *استرجع هذه دليله انك طبيعي*

HEPATOCELLULAR JAUNDICE

الذكري
انه ياتي نظري

1 ETIOLOGY

1. Acute: same causes of acute (fulminant) liver failure. *اشرح كل الاسباب*
2. Chronic: same causes of chronic liver failure. *ل.ق.و*
3. Familial hyperbilirubinemia: "mainly elevation of the indirect bilirubin"

- **Gilbert's disease:** "the only common one"
 - * It is due to a defect in the uptake & conjugation of bilirubin by the liver cells.
 - * v. mild jaundice: 3-3.5
 - * intermittent
 - * vvv. benign
 - * life expectancy 100%.
- **Crigler - Najjar syndrome:**
 - It is due to a deficiency of the conjugating enzyme (glucuronyl transferase).
 - It is of 2 types: Type I (severe deficiency), Type II (moderate deficiency).
- **Lucey - Driscoll syndrome:**
 - It is due to inhibition of the conjugating enzyme by a substance in the maternal serum.
- **Breast milk jaundice:**
 - It is due to inhibition of the conjugating enzyme by a substance in the breast milk.

v rare

oral
Hepatocellular disease
الى نزيه في
indirect only
not direct
↓
familial
حسب الاسباب فقط
↑
uptake conjugation
not excretion

2 Pathogenesis

3 CLINICAL PICTURE

1. Jaundice: is usually moderate (orange yellow).
2. Stools: pale.
3. Urine: dark.

4. Features of liver cell failure.

5. Features of the cause, e.g. "Familial hyperbilirubinemia":

- They are asymptomatic, or present with mild intermittent jaundice. (Gilbert)
- Only Crigler - Najjar is fatal.

عنوا 13 discuss LQ. if

4

INVESTIGATIONS

1. Serum bilirubin: increased (direct & indirect).
2. Stools: ↓ decreased **stercobilinogen**.
3. Urine: ↑ increased **urobilinogen**, bilirubin is present..

4. Investigations of liver failure, e.g. prolonged PT that is not corrected by parenteral vit. K.

5. Investigations for the cause, e.g. markers for viral hepatitis.

MCQ

عالم
تناقص
بيليروبين

normal ↑ Acute
Low ↑ chronic

IFLQ
اشباع
كبد
Liver fn tests
Albumin
Cholesterol

CLINICAL DIAGNOSIS OF JAUNDICE

A. PERSONAL HISTORY

1. Age:

- Children: hemolytic anemia, viral hepatitis.
- Adults: calcular obstruction, viral hepatitis.
- Old age: malignant obstruction.

2. Sex:

- Females: calcular obstruction.
- Males: malignant obstruction.

3. Special habits:

- Alcohol: liver cirrhosis leading to hepatocellular jaundice.

أي نوع ال
خلايا
عائقة تزداد
في الدم فتكاد المقروصه
تزيد
لكن هي مكان
مش بارقة
تصنع
فده يلقي ده
ويبقى ← normal

normal cholesterol
e defective esterification

B. PRESENT HISTORY

1. Onset:

- Acute: viral hepatitis, calcular obstruction.
- Gradual: cirrhosis, malignant obstruction.

2. Course:

- Progressive: cirrhosis, malignant obstruction.
- Regressive: viral hepatitis.
- Intermittent: calcular obstruction, hemolysis, Gilbert's disease, cancer ampulla of Vater.

3. Duration:

- Short: viral hepatitis.
- Long: cirrhosis (long duration will exclude malignancy).

4. Urine:

- Dark: obstructive jaundice, hepatocellular jaundice.
- Normal but darkens on standing: hemolytic jaundice.

5. Stools:

- Dark: hemolytic jaundice.
- Pale: hepatocellular jaundice.
- Pale + features of steatorrhea: obstructive jaundice.

6. Fever:

- Viral hepatitis: pre-icteric stage.
- Calcular obstruction: Charcot's fever (fever, rigors, jaundice & right hypochondrial pain).

7. Anorexia, nausea & vomiting:

- Viral hepatitis: pre-icteric stage.
- Calcular obstruction.

reflex pyloric spasm

8. Pain:

- Biliary colic: calcular obstruction.
- Epigastric pain radiating to the back: cancer head of pancreas.
- Dull aching pain in the right hypochondrium & epigastrium: viral hepatitis.
- Abdominal, back, & bone pains: hemolytic crisis.

qualitative mesenteric ischemia

oral

9. Pruritus:

- Obstructive jaundice & maybe hepatocellular.

10. Marked loss of weight:

- Malignancy.

حسب 10 كيلو
في الشهرين الماضيين

پایه ای epigastrium
و shoots خفشی از کتف راست
1. Pancreatic cancer head
2. Perforated Peptic ulcer
3. Dissecting Aortic aneurysm

C. PAST HISTORY

1. Biliary colic: calcular obstruction.
2. Blood transfusion & repeated injections: viral hepatitis.
3. Contact with cases of viral hepatitis, or frank past history of viral hepatitis.
4. Drug intake: intrahepatic cholestasis, hepatitis, hemolysis.

D. FAMILY HISTORY

- Hemolytic anemia.
- Familial hyperbilirubinemia.

OCPs
chlorpromazine

oral

jaundice زردی
hepatitis التهاب کبد
hemolysis
& methyl dopa
H.C.J ← hepatitis
H.J ← hemolysis

E. GENERAL EXAMINATION**1. Jaundice:**

- Mild: lemon yellow: hemolytic jaundice.
- Deep: olive green: obstructive jaundice.
- Moderate: orange yellow: hepatocellular jaundice.

2. Cachexia: in malignancy.**3. Manifestations of hemolytic anemia:**

leg ulcers & skin pigmentation.

4. Manifestations of obstructive jaundice:

scratch marks, Xanthomata & bradycardia.

5. Manifestations of liver cell failure:

e.g. palmar erythema & spider naevi.

6. Skin pigmentation & clubbing:

primary biliary cirrhosis.

7. Ecchymosis:

hepatocellular & obstructive jaundice. →

8. Oedema of lower limbs:

liver failure, & IVC obstruction by malignancy.

cancer head
if infiltrates
IVCالتهاب الكبد
Paroxysmal
night

WhiteKnightLove

F. ABDOMINAL EXAMINATION

1. Liver:

- **Viral** hepatitis: enlarged, **soft**, tender.
- Cirrhosis: sharp edge & shrunken.
- Obstructive jaundice: markedly enlarged.
- **Malignant** liver: **enlarged, hard, tender, irregular.**
- Hemolytic anemia: may be enlarged.

2. Gall bladder:

“Courvoisier's law”

- Calcular obstruction: small, tender, not palpable.
- Malignant obstruction: enlarged & palpable.

3. Splenomegaly:

- **Hemolytic** anemia. (esp thalassemia) (except SCA)
 autosplenectomy
- Liver cirrhosis & chronic active hepatitis.
- Viral hepatitis: sometimes.

4. Ascites:

- **Liver cell failure** & liver cirrhosis.
- Malignant **deposits** in the peritoneum.

5. Signs of portal hypertension:

e.g. dilated abdominal wall veins

- Liver cirrhosis.

Caput Medusae

6. Abdominal masses:

- Malignancy.

cell
 خلية
 عازقة
 B.le salts
 في intestine
 في الأمعاء
 White Knight Love

SERUM ENZYMES

37

1. Transaminases:

- Serum Glutamic Oxalacetic Transaminase (SGOT) = Aspartate Transaminase (AST).
- Serum Glutamic Pyruvic Transaminase (SGPT) = Alanine Transaminase (ALT).
- Normally: AST is present in: Liver, heart, kidney, skeletal muscles.
ALT is present in: Liver. *not > 40 units*
- In disease: These enzymes increase in DAMAGE of these organs (acute diseases).
- Conclusion: ALT is more specific for LIVER DISEASES than AST.

آل فانت
5000
6000 units
Chronic liver disease
100, 200 units

AST increases in: *hepatitis, fulminant liver failure*
active liver damage, acute myocardial infarction,
acute kidney affection, myopathy (severe cases)

ALT increases in: active liver disease, e.g. viral hepatitis.

2. Alkaline Phosphatase (ALP):

- Normally: It is formed mainly in: LIVER & BONE (3-13 King Armstrong units).
- In disease: It increases in: *lining of Bile ducts*

>30
5NT, GGT, ALP

• Marked increase in:

- LIVER DISEASE: *Space occupying lesions, Infiltrative disease of the liver.*
- BONE DISEASE.

13-30 • Moderate increase in:

- HEPATOCELLULAR DISEASES: *Hepatitis & Cirrhosis.*

How to confirm that increased ALP is due to LIVER DISEASE & not BONE DISEASE ??

- Associated increase in GGT.
- Associated increase in 5-nucleotidase.
- Abdominal ultrasonography.

3. Gamma Glutamyl Transpeptidase (GGT):

- It increases in: 1. Biliary obstruction. 2. Hepatocellular diseases (esp. Alcoholic hepatitis).

4. Serum 5-nucleotidase:

- It increases in: - Biliary obstruction.

Q - ALP = 50
A - marked increase
Q - *in liver*
A - liver or bone
Q - *in liver disease*
A - Biliary obstruction
Q - How differentiate from bone
A - Liver is +ve 5NT, GGT, ALP

not non specific

TESTS FOR PROTEINS

A. Chemical Separation of Plasma proteins:

- Total proteins: 6 – 8 gm / dl.
- Albumin: 4 – 5 gm / dl. *جرامان*
- Globulin: 2 – 3 gm / dl.
- A / G ratio: 2 / 1.

- CHRONIC LIVER DISEASE (e.g. liver cirrhosis):

- Albumin: Decreases.
- Globulin: Increases. *→ due to antigenemia of infection (Ag.)*
- A / G ratio: Reversed. *RES is activated synthesis*

- ACUTE LIVER DISEASE (e.g. viral hepatitis):

- Albumin: Is not affected (because it has a long half life).
- Globulin: Increases.

HYPOALBUMINEMIA is a marker of chronic liver disease

B. Electrophoresis of Plasma Proteins:

- Albumin: synthesized in the LIVER

It ↓ in CHRONIC Liver disease but not in acute liver disease.

- Alpha – 1 Globulin (e.g. Alpha – 1 antitrypsin): synthesized in the LIVER

It ↓ in CHRONIC Liver disease & in Alpha – 1 antitrypsin deficiency.

- Alpha – 2 Globulin & β – Globulin (e.g. LDL):

- They are LIPOPROTEINS that are excreted in bile.
- They increase in Obstructive jaundice.

- Gamma Globulins:

They increase in diseases that stimulate the RES:

- Acute & Chronic liver diseases.
- Autoimmune diseases.
- Infections & malignancy.

synthesized in the RES

liver spleen LNs

RES من الخلايا التي في
GG إلى إنتاج

lymphocytes

TESTS FOR FATS

- Normally: Blood cholesterol is 150 – 200 mg %.
- In disease:
 - o In Obstructive jaundice: Increased cholesterol, with normal esterification.
 - o In Liver failure: Normal cholesterol, with decreased esterification.

TESTS FOR CARBOHYDRATES

Glucose Tolerance Test:

of limited value in assessment of liver diseases

- In chronic liver failure: IGT & rarely secondary diabetes.
- In acute liver failure: Hypoglycemia.

no glycogenolysis → insulin resistance

PROTHROMBIN TIME (PT)

سأ مقصود

- Normally: Prothrombin time (PT) is 12 – 14 sec.
Prothrombin concentration (PC) is 100%. *INR = 1*
- In disease: PT is prolonged & PC is decreased in:

1. Obstructive jaundice & other causes of Vitamin K deficiency:

- Parental Vitamin K will correct the PT & PC.

2. Liver cell failure (acute & chronic):

- Parental Vitamin K will not correct the PT & PC.

لأنه في حالة فشل الكبد لا يمكن تصنيع فيتامين ك

CLINICAL IMPORTANCE OF PT

1. To assess the severity & prognosis of liver diseases.
2. Before any invasive technique or surgery. *must not be < 70%*
3. Follow up of oral anti – coagulants. *دواء مضاد تخثر فموي*

as in prosthetic valve MS & AF

لازم كل شهر متابعة التحليل

White Knight Love

*نيزن ↑
لور 5
لور 1
تجارب*

*2 1/2 INR ← هذا هو النورمال
القيمة*

TESTS FOR DETOXIFICATION

1. Bromosulphalein test (BSP):

- This dye is given in a dose of 5 mg / Kg IV.
- Normally: 90 % of the dye is cleared from the blood after 45 minutes.
This needs: normal hepatic blood flow, normal liver function,
patency of the biliary system.
- In disease: The dye will not be cleared from the blood after 45 minutes.
This occurs in: defective hepatic blood flow, abnormal liver function,
obstruction of the biliary system.

2. Indocyanin Green:

- It is safer & more specific than BSP.

ALPHA FETO-PROTEIN (AFP)

- Normally: It is absent in the serum, or very very low in the serum: < 20 ng / ml.

- In disease: It is present in the serum in the following conditions:

>500 ng

1. Marked elevation:

- Hepatocellular carcinoma (HCC).

20-500

2. Moderate elevation:

- Hepatitis & Cirrhosis.
- Pancreatitis.
- Malignancy: GIT, Ovary, Testis.

oral

Q: AFP ~!
A: Tumor marker for HCC

Q: specific or not
A: nonspecific

Q: but specific if >500

not sensitive

MCQ

Normal levels of AFP do not exclude HCC

& level of AFP doesn't correlate size of tumor

CLINICAL IMPORTANCE OF AFP

1. Follow up of cirrhotic patients for early detection of HCC.
2. Suspected cases of HCC: e.g. focal hepatic lesion.
3. After tumour resection:
 - Early: to confirm successful resection.
 - Late: to detect recurrence of the tumour.

for early detection

viral Hemochromatosis

malignant tumor
hemangiomas

INVESTIGATIONS FOR JAUNDICE

I. LABORATORY INVESTIGATIONS

A. Liver function tests

diagnose cause
↑ jaundice
DD.

1. Hemolytic jaundice:

- Serum: Increased serum bilirubin, mainly the indirect.
- Stools: ↑ Increased stercobilinogen.
- Urine: ↑ Increased urobilinogen, absent bilirubin.
- Other liver function tests: normal.

2. Obstructive jaundice:

- Serum: Increased serum bilirubin, mainly the direct.
- Stools: ↓ Decreased stercobilinogen.
- Urine: ↓ Decreased urobilinogen, bilirubin is present.
- Others liver function tests:
 - Enzymes: elevated enzymes (ALP, GGT, 5 – nucleotidase).
 - Prothrombin time: PT: prolonged & corrected by parenteral vitamin K.
 - Cholesterol: increased cholesterol with normal esterification.

3. Hepatocellular jaundice:

- Serum: Increased serum bilirubin, direct & indirect.
- Stools: ↓ Decreased stercobilinogen.
- Urine: ↑ Increased urobilinogen, bilirubin is present.
- Others liver function tests:
 - Enzymes: ALP: moderately elevated, AST & ALT: ↑ in acute cases.
 - Prothrombin time: PT: prolonged & not corrected by parenteral vitamin K.
 - Cholesterol: normal cholesterol with decreased esterification.
 - Proteins: decreased albumin & increased globulin in chronic cases.
↓ synthesis
↓ excretion

B. Blood Picture

- In Hemolytic jaundice: Features of hemolytic anemia. Normo Normo eRetics
- In Hepatocellular jaundice: Features of anemia due to liver cell failure. except thalassemia
- In obstructive : Macrocytic (as lipids ↑ & cholesterol deposits - wall of RBCs)

C. Serology

- In Hepatocellular jaundice: Hepatitis markers, antibodies for autoimmune hepatitis. (lupoid)
- In Obstructive jaundice: Anti-mitochondrial antibodies for: PBC.
- in hemolytic " : coomb's test (Abs against RBCs)

↑
anti liver kidney
mitochondrial Ab
ALP ↑
White Knight Love

II. IMAGING

1. Abdominal ultrasonography & CT

- Biliary system:** dilated intrahepatic biliary radicles in extrahepatic obstruction.
 - Gall bladder:** stones. (thick wall, contracted, tender on probing)
 - Liver:** cirrhosis, tumours, portal vein dilatation. = Normal = 12 mm if 13 mm dilated
 - Spleen:** splenomegaly.
 - Pancreas:** cancer head of pancreas.
 - Ascites.**
- ↓ = portal HTN
= Liver cirrhosis
= Hepatocellular jaundice

2. Visualization of the biliary system

- This is done mainly in: *extrahepatic obstruction.*
- This is done using: 3 techniques:

1. Endoscopic Retrograde Cholangio-Pancreatography (ERCP):

- Value:** visualization of the biliary system & pancreatic system.
- Indication:** obstructive jaundice, e.g. biliary stones or cancer head of pancreas.
- Therapeutic use:** stone removal by sphincterotomy. + put stent
- Complications:** cholangitis, pancreatitis.
- Method:** injection of a radio-opaque substance in the biliary system by a catheter introduced through a duodenoscope. & xray coils

2. Magnetic Resonance Cholangio-Pancreatography (MRCP):

- Accurate visualization of the biliary & pancreatic systems. *not therapeutic*

3. Percutaneous Transhepatic Cholangiography (PTC):

- Value:** visualization of the intrahepatic bile ducts.
- Indication:** obstruct. jaund. to differentiate intra- from extra- hepatic obstruction.
- Therapeutic use:** biliary drainage by a stent = PTD.
- Complications:** infection, hemorrhage. Bleeding tendency
- Method:** percutaneous injection of a radio-opaque substance into the intrahepatic bile ducts. V. invasive

III. LIVER BIOPSY

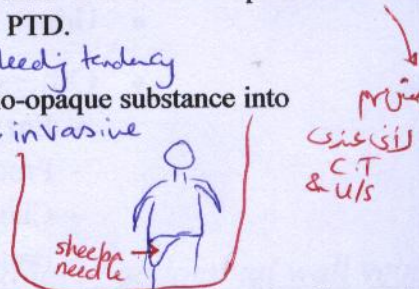
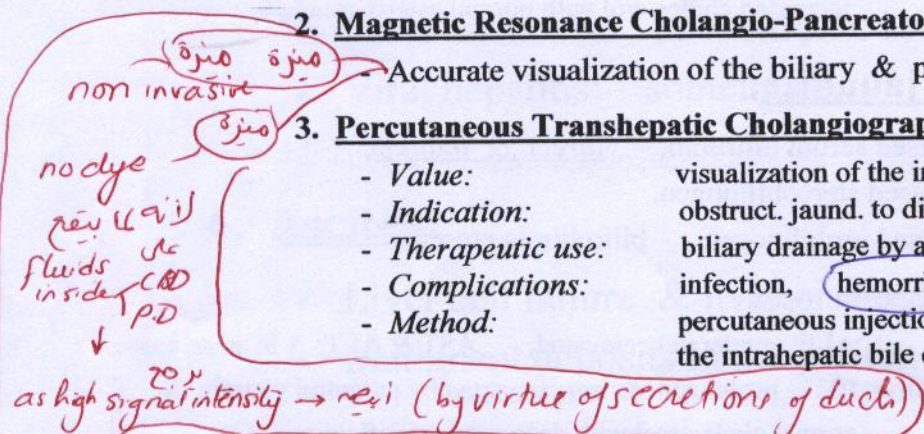
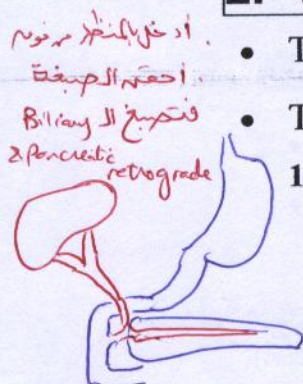
- Value:** diagnoses liver diseases & differentiates intra- from extra- hepatic obstruction.
- Precautions:** risky because of bleeding tendency & therefore PT, PC should be done first.

IV. LAPAROSCOPY

- It is used for visualization of the liver & GB & needle biopsy could be taken.

V. LAPAROTOMY

- It is rarely used now for the diagnosis.



B) General treatment:

rest in bed, + light nutrient diet.

C) Symptomatic treatment:

analgesics & antipyretics.

D) Treatment of complications:

antibiotics for secondary infection.

II. NEEDLE ASPIRATION

- It is better be guided by sonar or CT.
- It is indicated in cases not responding to medical treatment.

III. SURGICAL DRAINAGEindicated in:

- Cases not responding to medical treatment or needle aspiration.
- Huge abscess OR multiple abscesses.
- Secondary pyogenic infection.

FATTY LIVER**DEFINITION**

- Excessive deposition of fat in the liver. *fat wt is > 5% of Liver wt.*
- esp. TG : highest & fastest turnover among all fats*

ETIOLOGYI. Alcoholic steatosis & steatohepatitis.

II. Non alcoholic steatosis &

Non Alcoholic Steato Hepatitis (NASH)

- Obesity.
- Diabetes mellitus.
- Hyperlipidemia.
- Malnutrition.
- Drugs:

Corticosteroids, CCPs.

III. Acute fatty liver of pregnancy & Reye's syndrome.

IV. Idiopathic.

DIAGNOSIS

1. ASYMPTOMATIC: is the RULE, PAIN in the right hypochondrium is the EXCEPTION.
2. LIVER: enlarged, soft, smooth surface, may be tender. *(steatohepatitis)*
3. Acute liver failure: may occur in case of Acute fatty liver of pregnancy or Reye's syndrome.
4. Ultrasonography: bright hepatomegaly.
5. Liver biopsy: diagnostic.
6. Curcumin : NASH rarely

TREATMENT

1. Treatment of the cause. *eg: Proper DM control, stop alcohol*
2. Low fat diet.

Bright hepatomegaly
fatty — chronic hepatitis

↓ export
by ↓ apolipoproteins
* alcohol
* drugs & CCPs
* malnutrition

** obesity*
* hyperlipidemia
* DM

White Knight Love

HEPATIC SCHISTOSOMIASIS

ETIOLOGY

1. The ova:

- Schistosoma Mansoni:

Responsible for the majority of the cases because:

easy access to the liver.

- Schistosoma Haematobium:

Responsible for the minority of the cases because:

difficult access to the liver through vesico-mesenteric anastomosis.

2. Distribution:

more common in Delta region where S. mansoni is prevalent.

3. Incidence:

more common in males, usually between 10 & 40 years.

PATHOGENESIS

1. The granuloma:

- They reach the liver via the portal system to induce granuloma formation in the portal tracts.
- The granuloma is formed of: ova surrounded by lymphocytes, macrophages & eosinophils.

2. Periportal fibrosis:

- The granuloma will later fibrose leading to: "periportal fibrosis" around the portal veins.

3. Portal Hypertension:

- This results from periportal fibrosis.

4. NO LIVER CELL FAILURE:

- In pure Schistosomiasis: There is ONLY FIBROSIS, no cirrhosis, & no liver cell failure.
- In mixed pathology: many cases of Hepatic Schistosomiasis are associated with viral hepatitis, especially HCV, (mixed pathology) resulting in: cirrhosis & liver cell failure.

CLINICAL PICTURE

Symptoms

- Early cases: asymptomatic OR symptoms of intestinal bilharziasis.
- Late cases:

1. Symptoms of Portal Hypertension:
2. Symptoms of Liver Cell Failure:

e.g. bleeding oesophageal varices.

ABSENT in pure Hepatic Schistosomiasis.

PRESENT in mixed pathology.

General Signs

1. Signs of Liver Cell Failure:

ABSENT in pure Hepatic Schistosomiasis.

PRESENT in mixed pathology.

2. Signs of nutritional deficiency:

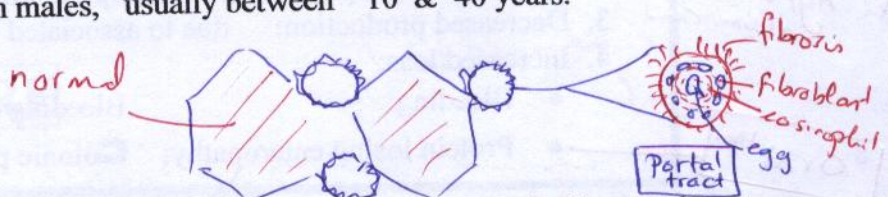
3. Signs of infantilism:

4. Signs of bilharziasis in other areas:

Bilharzial cor pulmonale. : signs of Pulm. HTN

lung 3! hematobium!
cor pulmonale 3! mansoni 3!
= failure to develop 2ry sexual characters after puberty

if mixed pathology (LCF) disturbed hormones
no proteins
White Knight Love



عائین
تووع
لا كانوا بسحوا
mes
fistulae
عند
باصي ريت طلع

Fever
jaundice
Bleeding tendency

Palmar erythema
gynecomastia

Abdominal Signs

1. Hepatomegaly: enlarged liver in early cases & shrunken liver in late cases.
2. Splenomegaly: enlarged spleen due to portal hypertension & may reach a HUGE size.
3. Ascites: may develop late due to associated HYPOALBUMINEMIA.
4. Signs of chronic increase in the intra-abdominal pressure: (organomegaly)
 - Subcostal angle: widening.
 - Umbilicus: everted, shifted downwards, $\pm$ umbilical hernia.
 - Divarication of the recti.
5. Dilated abdominal veins (Caput medusae) & venous hum may be present.

CAUSES OF ASSOCIATED HYPOALBUMINEMIA

1. Decreased intake: nutritional deficiency due to poverty.
2. Decreased absorption: malabsorption due to intestinal congestion.
3. Decreased production: due to associated cirrhosis in mixed pathology.
4. Increased loss:
 - Bleeding: Bleeding oesophageal varices.
 - Protein losing enteropathy: Colonic polyps & Congestive enteropathy.

CLINICAL STAGES OF HEPATIC SCHISTOSOMIASIS

1. Hepatomegaly.
2. Hepatosplenomegaly.
3. Shrunken liver & splenomegaly.
4. Shrunken liver, splenomegaly & ascites.

INVESTIGATIONS

1. Investigations for diagnosis of Schistosomiasis:

- Stool & urine analysis: for Schistosoma ova.
- Sigmoidoscopy & biopsy: for Schistosoma ova. rectal snip = heavily ova
- Serology & skin tests: for Schistosoma antibodies.
- Liver biopsy: shows the typical granuloma & periportal fibrosis.

2. Investigations for diagnosis of Portal Hypertension:

e.g. upper GI endoscopy: for oesophageal varices.

3. Investigations for Liver Function Tests:

- NORMAL in pure Hepatic Schistosomiasis.
- AFFECTED in mixed pathology.
- Hypoalbuminemia may be present (see the above table).

4. Investigations for Liver Imaging:

Abdominal ultrasonography & CT.

non specific: dil portal vein: > 1.2 cm
v. specific: portal tract thickening = Periportal fibrosis

TREATMENT

1. Treatment of active Schistosomal infection: anti-bilharzial drugs especially Praziquantel.
2. Treatment of Portal Hypertension. *eg. injectate Sclerotherapy*
3. Treatment of Liver cell failure: *in cases of mixed pathology. eg. H Hepatic encephalopathy*
4. Treatment of associated viral hepatitis: *in cases of mixed pathology. eg.*

INTESTINAL BILHARZIASIS

ETIOLOGY

- It is due to *S. Mansoni* ova which live in the mesenteric plexus of veins.
- It is very rarely due to *S. Haematobium* which live in the vesical plexus of veins due to vesicomesenteric anastomosis.

CLINICAL PICTURE

1. Asymptomatic:

most of the cases.

2. Intestinal manifestations:

- It occurs in cases of: polyps or ulcers in the colon. *→ typical β granuloma*
- It presents mainly with: bleeding per rectum. *fresh (curr)*
- It presents also with: chronic diarrhea & dysentery. *→ toxins of ova*
- It is associated with: a pericolic mass in the left iliac fossa. *= inflammatory*

3. Extra – intestinal manifestations:

- Anemia of chronic disease. *(infect, inflammation)*
- Clubbing. *Fe deficiency*
- Hepatic Schistosomiasis.
- Bilharzial cor pulmonale.

"Complications"

defecation str. & painful incomplete evacuation
defecation → persistent desire to defecate

(Colorectal but more rectal)

DD:

• Spastic Colon

• Fecal

• Diverticular disease

- TB
- Crohn's
- Cancer colon
- Sigmoid volvulus

INVESTIGATIONS

1. Investigations for diagnosis of Schistosomiasis:

- **S**tool & urine analysis: for *Schistosoma* ova.
- **S**igmoidoscopy & biopsy: for *Schistosoma* ova (*appear in biopsy from polyps & ulcers*).
- **S**erology & skin tests: for *Schistosoma* antibodies.
- **B**arium enema: for *Schistosoma* polyps in the colon.
- **B**lood picture: for anemia & eosinophilia. *→ if ACD: micro or Normo micro WBCs*

2. Investigations for diagnosis of complications:

e.g. liver imaging for hepatic schistosomiasis.

Lung = dumbbell shaped pulm. trunk (P-H.)

TREATMENT

1. Treatment of active Schistosomal infection: anti-bilharzial drugs especially Praziquantel.
2. Treatment of polyps: endoscopic polypectomy.
3. Symptomatic treatment: e.g. treatment of anemia, antidiarrheal drugs for diarrhea.

Polyp is never precancerous
Familial polyposis
هو اللى
precancerous

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LIVER ENLARGEMENT

I. LIVER INFLAMMATION

نادراً
نظري
ذاكر

1. Viral:

- Viral hepatitis: A, B, C, D, E.
- Other viruses causing hepatitis: EBV, CMV, HSV, Yellow fever.

2. Bacterial:

- Brucellosis.
- Typhoid.
- TB (miliary).
- Syphilis.
- Pyaemic abscesses.

3. Protozoal:

- Amoebic liver abscess.
- Malaria.
- Kala - azar.

4. Helminthic:

- Schistosomiasis.
- Hydatid cyst.
- Fasciola.

II. LIVER CONGESTION

- Right ventricular failure.
- Tricuspid valve disease (regurge or stenosis).
- Pericardial disease (effusion or constrictive pericarditis).
- High IVC obstruction.
- Budd - Chiari syndrome.
- Veno - occlusive disease.

III. LIVER CIRRHOSIS

- Cardiac & Biliary cirrhosis.
- Early stages of other causes of cirrhosis.

IV. LIVER TUMOURS

- Primary: *Benign adenoma* OR *malignant HCC*.
- Secondaries.

V. OBSTRUCTIVE JAUNDICE

- Especially extrahepatic obstruction.

VI. BLOOD DISEASES

- Anemia: *hemolytic* & *megaloblastic*.
- Polycythemia vera.
- Blood malignancies: *leukemia* & *lymphoma*.

VII. METABOLIC DISEASES

- Fatty liver.
- Hemochromatosis.
- Wilson's disease.
- Amyloidosis.
- Lipid storage diseases: *e.g. Gaucher's disease, Neimann – Pick disease.*
- Glycogen storage diseases: *e.g. Von Gierke's disease.*

VIII. MISCELLANEOUS

- Collagen diseases: *e.g. SLE.*
- Granulomatous diseases: *e.g. Sarcoidosis.*
- Inflammatory bowel disease: *Ulcerative colitis, Crohn's disease.*

LIPID STORAGE DISEASES

- Hereditary disorders characterized by abnormal accumulation of lipids in the tissues.
- They are rare disorders affecting Jewish children.

1. Gaucher's disease:

- o Huge splenomegaly & moderate hepatomegaly.
- o Skin: pigmentation.
- o Bone: pains & fractures.
- o Bone marrow: characteristic GAUCHER CELLS.

2. Neimann – Pick disease:

- o Moderate splenomegaly & huge hepatomegaly.
- o Blindness, deafness, mental retardation.
- o Bone marrow: characteristic NEIMANN – PICK CELLS.

GLYCOGEN STORAGE DISEASES

- Hereditary disorders characterized by abnormal accumulation of glycogen in tissues.

Von Gierke's disease:

- o Hepatomegaly.
- o Cardiomegaly.
- o Hypoglycemia.
- o Short stature.

DRUG - INUCED LIVER DISEASE

- | | |
|-----------------------|--|
| 1. Acute hepatitis: | alcohol, INH, halothane, paracetamol. |
| 2. Chronic hepatitis: | alcohol, INH, methyl dopa. |
| ③ Cirrhosis: | alcohol, INH, methyl dopa, amiodarone, methotrexate. |
| ④ Cholestasis: | CCPs, Chlorpromazine. |
| 5. Fatty liver: | Corticosteroids, CCPs. |
| 6. Tumours: | CCPs, Androgen. |

HEPATITIS

نظري

A. ACUTE HEPATITIS:

< 6 Months

1. Infections:

- Viral hepatitis: A, B, C, D, E.
- Other viruses causing hepatitis: EBV, CMV, HSV, Yellow fever.

2. Drugs & toxins:

- Alcohol, Halothane, INH, Paracetamol.

3. Metabolic:

- Wilson's disease. (also causes liver cirrhosis)

الفرق
مكتلة
وحدة

B. CHRONIC HEPATITIS:

> 6 Months

1. Chronic active hepatitis.

carries virus &
v. severe pathology
symptomatic

2. Chronic lobular hepatitis.

carries virus but
v. mild inflammation
→ not clinically manifested

3. Chronic persistent hepatitis.

healthy carrier (asymptomatic)

ACUTE VIRAL HEPATITIS

1

ETIOLOGY

- Viral hepatitis: A, B, C, D, E.
- Other viruses causing hepatitis: EBV, CMV, HSV, Yellow fever.

Hepatitis A Virus (HAV):

- Infection: Fecal-oral.
- Incubation period: 2-6 weeks.
- Incidence: Sporadic OR epidemic.
- Age: Children & young adults.
- Acute stage: Usually mild.
- After hepatitis (sequelae): No chronicity & No relation to HCC.
- Prophylaxis: Vaccination OR immunoglobulins.
- Genome: RNA.

Hepatitis B Virus (HBV):

- Infection: Parental, Sexual, Vertical (from mother to child).
- Incubation period: 1-6 months.
- Incidence: Sporadic.
- Age: Any age.
- Acute stage: Usually severe.
- After hepatitis (sequelae): Chronicity & HCC.
- Prophylaxis: Vaccination OR specific immunoglobulins.
- Genome: DNA.

⑤ HCV

هو أكثر واحد يعل
HCC

② HAV as HEV

but: 2 differences

1 severe in E

if pregnant

2 no vaccine in E

⑦ HBV as HDV

but: 2 differences

1 HCC / no HCC

2 DNA / RNA

⑥ E & A

خذ

خذ

خذ

خذ

④ Acute stage
⑧ severe
⑨ mild
⑤ fulminant
- HBV
HEV in pregnant
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نظري

- Diagnosis
or - Management

Oral 1000

بالذات
etiology

etiology

لا تسأل نظري
أكن في سنو

* قواعد *

① RNA كلب

② A, E → fecal-oral

③ RNA كلب

④ Acute stage

⑤ fulminant

⑥ E & A

⑦ HBV as HDV

⑧ severe

⑨ mild

⑤ fulminant

In Egypt 25% HCV: كل 4 أشخاص واحد عنده مع التناؤل المنزلي

doubtful ?? sexual vertically

Hepatitis C Virus (HCV):

- Infection: Parental, Community acquired (unknown etiology).
- Incubation period: 1 – 6 months.
- Incidence: Sporadic.
- Age: Any age, but more commonly in adults.
- Acute stage: Usually mild or unnoticed.
- After hepatitis (sequelae): Chronicity & HCC.
- Prophylaxis: Hygiene including supervision of blood donation.
- Genome: RNA.

مش عارضه انتقال اراي

تلاقى مفضي history

الحملات انه فيه نقل دم او غيره

فما تلاقى واحد عنده virus حرق من منه يتلوي لانه مش كل الطرق في الانتقال معرونة

ما يسالك يتقل Sexual فالعديس اجابة

Hepatitis D Virus (HDV):

- Infection: Parental, Sexual, Vertical (from mother to child).

HDV is a weak incomplete virus "Delta agent" depending on HBsAg, Therefore:

- It infects only those with HBV, & there are 2 forms of infection:

1. Co-infection: simultaneous infection with HBV & HDV.
 - This gives the picture of acute hepatitis which may be fulminant.
2. Super-infection: super-infection of a chronic HBV carrier with HDV.
 - This causes activation of hepatitis in a previously stable patient.

- Incubation period: 1 – 6 months.
- Incidence: Sporadic.
- Age: Any age.
- Acute stage: Usually severe.
- After hepatitis (sequelae): Chronicity.
- Prophylaxis: Vaccination against HBV.
- Genome: RNA.

HDV not enter body except if has HBV but HDV when enters on top of HBV it doesn't increase incidence of HCC of HBV

NO HCC

فارقين B

Hepatitis E Virus (HEV):

- Infection: Fecal – oral.
- Incubation period: 2 – 6 weeks.
- Incidence: Sporadic OR epidemic.
- Age: Children & young adults.
- Acute stage: Usually mild except in pregnant females.
- After hepatitis (sequelae): No chronicity & No relation to HCC.
- Prophylaxis: Hygiene & immunoglobulins.
- Genome: RNA.

not vaccine

فارقين A

Oral مية في بيرة

دخول مع بيرة

دخول في D

HBV يدخل HBV بحمل حبة من 1 Acute stage وحف الجسم ينشط من virus

او 2 Chronic stage لا ال virus مفعشش مفعشش (asymptomatic) stable دخول مفعشش HDV مفعشش stable Active

2 CLINICAL PICTURE

A. Anicteric Hepatitis:

- They are mild cases with:
- They present with:
- They are usually missed in diagnosis.

no jaundice.

influenza-like OR typhoid-like symptoms.

3 stages

B. Icteric Hepatitis:

1. Pre-icteric stage:

(3 days - 2 weeks)

Symptoms

1. Fever: acute onset.
2. Liver: dull aching pain in the right hypochondrium & epigastrium.
3. GIT: anorexia is marked especially to cigarettes, + nausea + vomiting.

Signs

1. Fever: with relative bradycardia.
2. Liver: enlarged, tender, soft.
3. Skin: non specific rash.

2. Icteric stage:

(1 - 4 weeks)

Symptoms

1. Fever: drop of fever & improvement of the general condition.
2. Jaundice: Dark urine occurs first followed by pale stools followed by jaundice.
3. GIT: anorexia + nausea + vomiting markedly improve OR disappear.

Signs

1. Fever: absent.
2. Liver: enlarged, tender, soft.
3. Spleen: slightly enlarged in 20 % of cases.
4. LN: slightly enlarged cervical LN in 10 % of cases.

3. Convalescence stage: post-icteric

Symptoms

- Gradually improve, then disappear.

Signs

- Gradually improve, then disappear.

Complete recovery of the liver (clinical, biochemical & histological) may take 6 months

3 SEQUELAE

A. COMPLETE RECOVERY

- In most cases of: HAV & HEV.
- In many cases of: HBV & HDV.
- In few cases of: HCV.

occurs in:

acute

Oral از اعتقاد آنه تحول هپاتوسلولاری obstructive

- ALP jumps to >30
- deepening of jaundice
- prolongation $\rightarrow$ to 28 w (7H)

72

B. COMPLICATIONS

I. Hepatic complications

1. Relapses:

one of 2 forms:

- **Clinical** relapse: the original attack recurs & jaundice reappears.
- **Biochemical** relapse: serum bilirubin & enzymes rise once again.

2. cholestatic Hepatitis:

Bile salts retained

especially with HAV

- Prolonged cholestasis: pruritus, jaundice, elevated ALP
- Persistent jaundice: for 8 – 28 weeks, followed by complete recovery.

3. Fulminant Hepatitis:

بسیار

massive hepatic necrosis

- Presents with: features of acute liver failure BUT is rare.

4. Post - Hepatitis Syndrome:

Transaminitis

- Presents with: fatigue, anorexia, pain in the right hypochondrium.
- Investigations: all are normal EXCEPT mild rise of transaminases.

5. Chronic sequelae:

Only in HBV, HCV, HDV

Not A, E

- **Chronic hepatitis.** (symptomatic)
- **Cirrhosis:** Post - Hepatitis Cirrhosis.
- **Cancer:** Hepatocellular Carcinoma. C, B (Not D)
- **Carrier state.** (asymptomatic)

II. Extra - hepatic complications

due to Circulating Immune Complexes

(also in Infective endocarditis)

- **Arthritis.**
- **Aplastic anemia.** (HBV, HCV)
- **Guillain - Barré syndrome.**
- **Glomerulonephritis** (only with HBV & HCV).
- **PAN & Cryoglobulinemia** (only with HBV & HCV).

$\rightarrow$ Immune complexes deposited in vessel wall $\rightarrow$ Vasculitis

هپاتیت مزمن

- auto immune disease $\bar{e}$ production of Abs only active in cold weather
- presents clinically by - joint: arthralgia - arthritides
- Kidney: GN
- skin: vasculitic skin lesion

• Diagnosis:

serology: **cryoglobulins** & normal albumin. $\downarrow$ ESR

4 INVESTIGATIONS

1. Liver function tests:

Hepatocellular J.

- Serum bilirubin: increased, **direct & indirect.**
- Transaminases: increased **markedly.**
- ALP: increased **moderately.**
- Proteins: increased globulin (Gamma globulins) & normal albumin.

RES hyperplasia

markedly if obstructive J

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2. Liver imaging:

“ Abdominal ultrasonography ”

- Liver: **Hepatomegaly.**
- Spleen: **Splenomegaly** in 20 % of cases. *RES hyperplasia*

3. Urine analysis:

- Colour: **dark & frothy** due to the presence of **Bilirubin** & **Bile salts**.
- **Urobilinogen:** increased.
- Others: Slight **proteinuria** & **hematuria** may be present due to GN. *Casts*

4. Stool analysis:

- Colour: pale with steatorrhea.
- **Stercobilinogen:** decreased.

5. Blood picture:

- **Leucopenia** with relative lymphocytosis.

6. HEPATITIS MARKERS:

A. Hepatitis “ A ” Markers:

- Anti - HAV antibodies: **IgM** denotes recent infection, **IgG** denotes old infection.

B. Hepatitis “ B ” Markers:

1. HBs Ag (Hepatitis B surface antigen):

- **Appears** 6 weeks after infection & **disappears** after 3 months.
- Persistence more than 6 months indicates either: **carrier state** or **chronic state**.

2. Anti - HBs antibodies:

- Appear after 3 months & persist.
- They indicate either: **recovery** & **immunity**.

3. HBcAg (Hepatitis B core antigen):

- It is not detected in the blood, it is only detected in liver biopsy.

4. Anti - HBc antibodies:

- **IgM:** Chronic active B hepatitis.
- **IgG:** Carrier OR old infection.

5. HBeAg:

- Its presence: High infectivity.
- Its persistence more than 3 months: Chronicity.

6. Anti - HBe antibodies:

- Its presence: Low infectivity.

7. PCR for HBV DNA:

- It is the most sensitive index for viral replication.

2. Liver imaging:

"Abdominal ultrasonography"

- Liver:
- Spleen:

Hepatomegaly.

Splenomegaly in 20 % of cases.

RES hyperplasia

3. Urine analysis:

- Colour:
- Urobilinogen:
- Others:

dark & frothy due to the presence of **Bilirubin** & **Bile salts**.
increased.

Slight **proteinuria** & **hematuria** may be present due to GN.

Casts

4. Stool analysis:

- Colour:
- Stercobilinogen:

pale with steatorrhea.

decreased.

5. Blood picture:

- Leucopenia with relative lymphocytosis.

virus لا سبب
++ RES

6. HEPATITIS MARKERS:

A. Hepatitis "A" Markers:

- Anti - HAV antibodies:

IgM denotes recent infection,
Acute

IgG denotes old infection.

الغلبة

أهم حاجة
في حصة
Hepatitis

B. Hepatitis "B" Markers:

1. HBs Ag (Hepatitis B surface antigen):

- Appears 6 weeks after infection & disappears after 3 months. → Acute infect.
- Persistence more than 6 months indicates either: **carrier state** or **chronic state**.
asymptomatic symptomatic

2. Anti - HBs antibodies:

- Appear after 3 months & persist.
- They indicate either: **recovery** & **immunity**.

3. HBcAg (Hepatitis B core antigen):

- It is not detected in the blood, it is only detected in liver biopsy.

4. Anti - HBc antibodies:

- **IgM:** Chronic active B hepatitis.
- **IgG:** Carrier OR old infection.

5. HBeAg:

- Its presence: High infectivity.
- Its persistence more than 3 months: Chronicity.

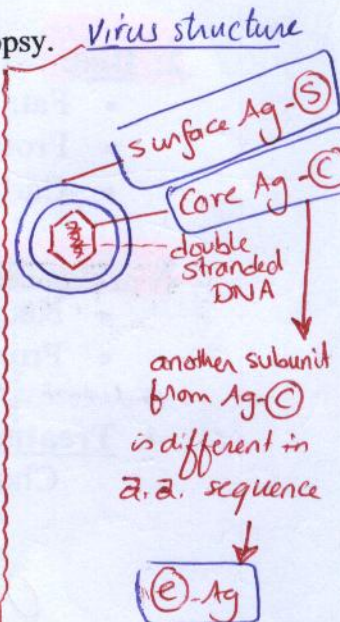
6. Anti - HBe antibodies:

- Its presence: Low infectivity.

7. PCR for HBV DNA:

- It is the most **sensitive** index for viral **replication**.

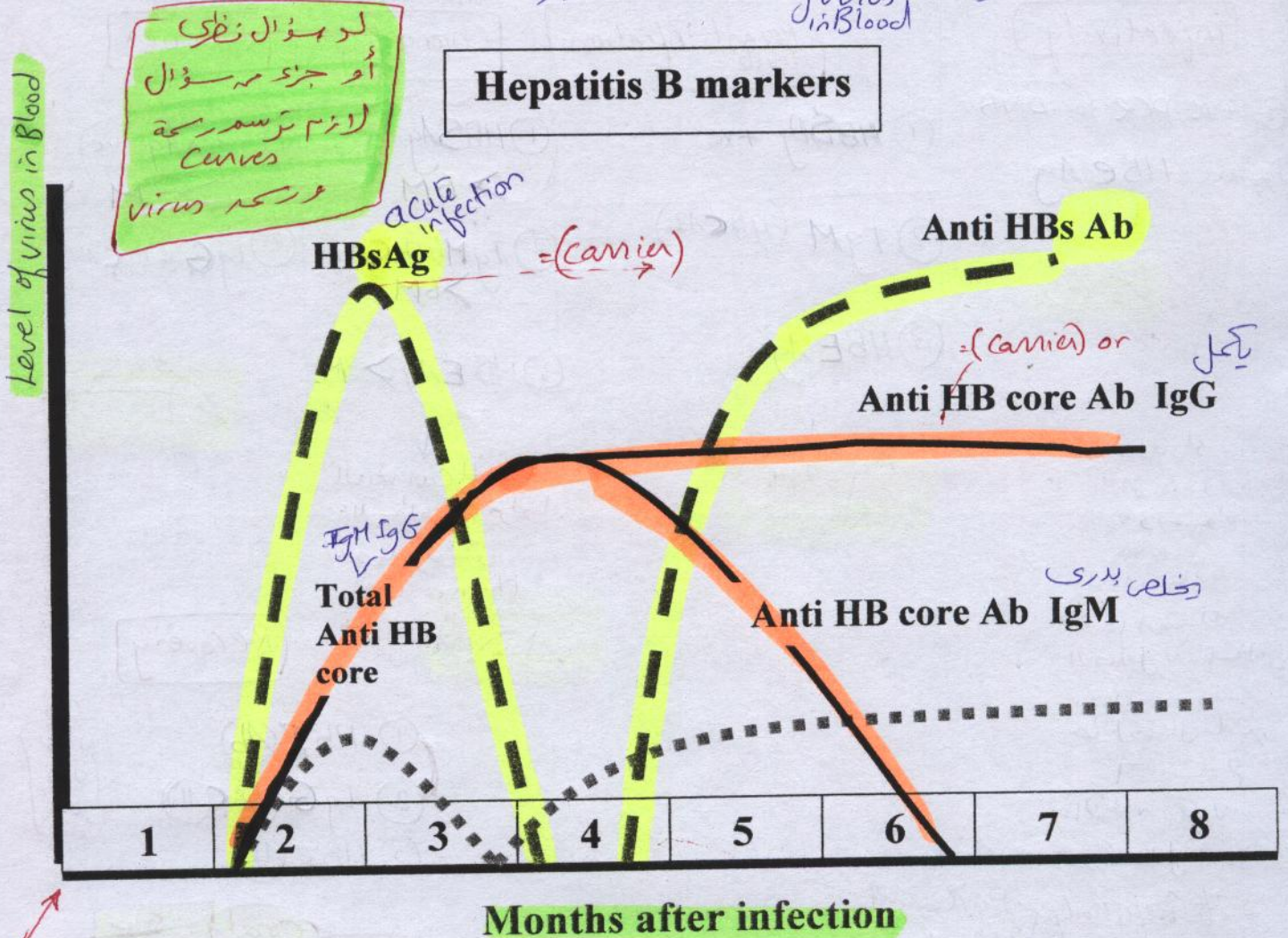
= infectivity



S-Ag	S-Ab
C-Ag	C-Ab
e-Ag	e-Ab

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نشوف level of virus in blood على حركاته دور بعد، بعدوى



..... = HBeAg & Anti HBeAb

لو تركته من غير علاج خالص

① HB SAg → appears after 6w & disappears after 3M → if persistent > 6M = chronicity or carrier (symptomatic) or carrier (asymptomatic)

HB SAb → appears in 3-3½ M & persists for life → if +ve = recovery or immune (old infection) or immune (vaccinated)

② HB C Ag → never in blood (only in liver tissue) Biopsy

Oral HB SAg → نظر انتي؟ فانت تجاوبه بقولك جيب

HB C Ag → نظر انتي بقولك مبيظهرش

HB C Ab → vaccine (HB C Ag) → +ve HB C Ab → recovery (old infection) or immune (vaccinated)

Sherif EL Hawary, MD

HB C Ab (Total) → IgG → appear 6w & persists → if +ve = recovery (old infection) or carrier

IgM → " 6w & disappears 6M → if +ve = acute infection

③ HBe Ag → 6w & 3M → high infectivity & Acute → if > 3M → chronic

HBe Ab → 3M & persists → if +ve → low infectivity (↓ replication) Recovery old infection

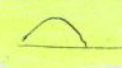
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Evidences that prove

تجسبات

infectivity

- ① +ve PCR for DNA
- ② +ve HBeAg



لجنى لو
اشكيت
نوم واحد
عنه
لوعايز تتأكد
هتعمل نفسك
التحليل لك هتأكد
انه صاخر وقت
موتيل عبال ما ظهر
7 اسبوع
فالاحسن تعال له
كهو التحليل

على PCR
في infectivity
في HBeAg

NB① Seroconversion

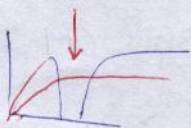
عند تحول
مرحلة لا تجد فيه
window phase



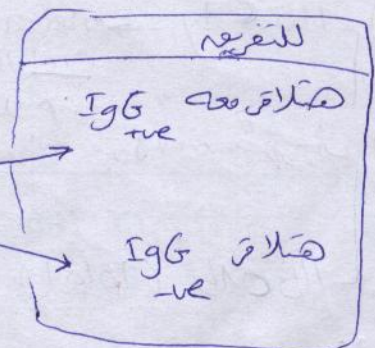
طب الحل : تجسبات ازاى ؟ بار IgM اذى لو

أحسن
more sensitive
CoreAb

NB② if HBsAb is +ve ⇒ either/or

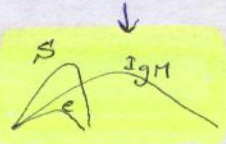


Recovery
Immune



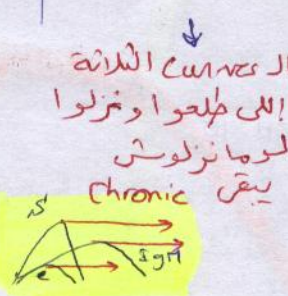
Recent infection Acute

- ① HBsAg +ve
- ② IgM (HBeAb)
- ③ HBeAg



Chronicity

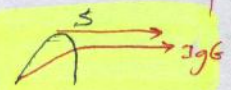
- ① HBsAg +ve > 6M
- ② IgM (HBeAb) > 6M
- ③ HBeAg > 3M



المرحلة الثالثة
الى ملعو او نزلوا
لوما نزلوش
يقر Chronic

asymptomatic Carrier

- ① HBsAg +ve > 6M
- ② IgG (HBeAb)



Recovery

- ① HBsAb
- ② IgG (HBeAb)
- ③ HBeAb

الى
مقلوب
حون
العمر

Oral

اذاى تغري
Carrier or recovery
بالعمل

تقول له

Carrier ← HBsAg
recovery ← HBsAb

C. Hepatitis "C" Markers:

1. Anti - HCV antibodies:

- Detected after 6 weeks of infection.

by ELISA₃ OR RIBA₄

3rd generation

4th generation

Enzyme Linked
Immuno Sorbent
Assay

Radio
Immuno
Block Assay

2. PCR for HCV RNA:

- Detected after 2 weeks of infection.

لواتسك وعائز تعرف عندك (C) ولا لا
منه فتعرف ان بعد اسبوعين
وكان PCR

D. Hepatitis "D" Markers:

1. Anti - HDV antibodies:

- IgM: recent infection.
- IgG: old infection.

2. PCR for HDV RNA:

تكرره ملاين المرات → +ve = multiplication

3. HBsAG is positive.

as HBV must be present to allow (D) to enter

CO infection super infection

E. Hepatitis "E" Markers:

Anti - HEV antibodies:

- IgM: recent infection.
- IgG: old infection.

TREATMENT

1. Bed rest:

- The patient is:
- The liver is:
- The serum bilirubin is:

until:

symptom-free.

no longer tender.

less than 1.5 mg / dl.

دامتي تقول
له عيني
تخون

2. Diet:

- Fat:
- Protein:
- Carbohydrates:

restricted because they aggravate nausea.

given freely BUT should be restricted if liver failure develops

High carbohydrate diet.
energy to liver

fulminant
B & D
or E in pregnant (rare)

3. Symptomatic treatment:

- Nausea:
- Pruritus:

domperidone (anti-emetic).

cholestyramine.

Bile salt
sequesterant
بيل سالت
سكويرنت

Cholestyramine
نارست اكلو لوراجيل

4. Treatment of complications:

Cholestatic hepatitis:

corticosteroids. to relieve edema in obstruct Bile ducts

Oral uses of Cortisol - Hepatitis < Acute

6 PROPHYLAXIS

1. Proper hygienic measures.

2. Immunoglobulins: given to exposed persons as soon as possible after exposure.

(Passive)

(Active)

3. HEPATITIS B VACCINE:

- Types: plasma – derived (from HBV carriers), or yeast – derived (recombinant).
- Efficacy: effective response in 95 % of persons.
- Indication: given to groups at **High Risk** for **HBV** infection including:
 - Health professionals: doctors, nurses, etc.....
 - Hemodialysis patients. *قبل نقل، ركي*
 - Hemophiliacs & Hemolytic anemia patients. *نقل دم كثير*
 - Homosexuals & IV drug abusers. *SCA Thalassemia*
 - Babies born to HBsAg – positive mothers.

المستقر
0, 1, 6, 5, 1 or 5
Months years

"فَفَهَّمْنَاهَا سُلَيْمَانَ وَكُلًّا آتَيْنَاهَا حُكْمًا وَعِلْمًا"

CHRONIC ACTIVE HEPATITIS

1. Definition

> 6M

chronic lobular
mild pathology
asymptomatic

من أهم
الدروس
النظري في ركي

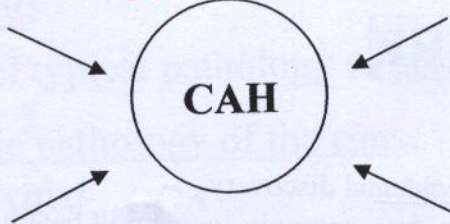
2. ETIOLOGY

1. Viral Hepatitis: B, C, D. (never A, E)
2. Auto – immune: Lupoid hepatitis.
3. Drugs & toxins: Alcohol, INH, methyl dopa. anti HTN anti TB
4. Metabolic: Wilson's disease, alpha – 1 antitrypsin deficiency.

(never Paracetamol)
↓
acute

في ركي
- viral esp C
- lupoid
Viral Hepatitis

ORAI
عنه يجي cirrhosis
في سن صغير



Drugs & Toxins

Auto immune

Metabolic

Investigation ۱

= cells

- C.T. separating
PZ from
hepatic
lobule

fuji ←
portal & Central
(portal Central)
gr
portal & portal
r
Centro Central

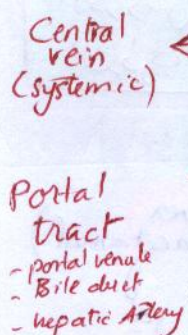
- The previous features plus:**

22

علامة
أكيدة
في
العلاج
Carrhosis

necrosis +++ → stellate cells → TGFβ

(Chronic Hepatitis $\rightarrow$ is pre cirrhotic)



CAH (mild)

CAH (severe)

R = Rosettes
BN = Bridging necrosis

4 CLINICAL PICTURE

Symptoms

- Asymptomatic: accidental discovery.
- Non – specific symptoms: fatigue, anorexia, general ill health.
- Hepatic symptoms: pain in the Rt hypochondrium, jaundice, liver failure lately.
- Extra-Hepatic symptoms: symptoms of cryoglobulinemia, GN, arthritis, etc.... may be present especially in autoimmune cases.

Signs

Hepatic signs:

- **Liver:** enlarged & firm.
- **Spleen:** enlarged. *RES hyperplasia in viral autoimmune*
- **Signs of complications:**
 - Liver cirrhosis: *shrunken* liver.
 - Liver cell failure & Portal hypertension: are late.
 - May be: *HCC.*

ORAL

التهاب مزمن

Extra-Hepatic signs:

- Signs of cryoglobulinemia, **GN**, arthritis, etc..... may be present especially in autoimmune cases. *more common in lupoid*
(يعني ما باليك هي مشورة مع الviral فما بالك لو السبب من الviral)

5 INVESTIGATIONS

most imp
in Biopsy

التهاب مزمن

1. Liver function tests: (hepato cellular J)

- Serum bilirubin: increased, *direct & indirect.*
- Transaminases: increased. *moderately* (الزيادة معتدلة)
- ALP: increased moderately. (*markedly only = OJ*)
- Proteins: increased globulin & decreased albumin.
- Prothrombin time: prolonged. *not corrected by parenteral vitk* *RES hyperplasia* *hypoalbuminemia is marker of chronic liver disease*

2. Liver imaging:

- Liver: Bright hepatomegaly.
- Spleen: Splenomegaly.

Bright = *Chronic hepatitis*
Fatty

3. Liver biopsy:

A) General typical pathology of chronic hepatitis.

B) Specific pathology of the **cause:** e.g.

- HBV: **GGA** & Positive orcein stain.
- HCV: Lymphocyte deposits & Fatty infiltration (**STEATOSIS**).

as cirrhosis

استئخ بؤع الالتهابي المزمن

4. Investigations for the cause: e.g.

- Hepatitis markers: for **B, C, D.**
- Auto-antibodies: for autoimmune hepatitis (**ANA, ASMA, ALKMA**).

Viral
autoimm.

استئخ

v. non specific

v. specific

Anti nuclear Ab

Anti Smooth Muscle Ab

Anti Liver Kidney Microsomal Ab

ORAL

eg. PBC → anti Mitochondria
GBS → Anti glyco lipid
SLE → Anti double stranded

White Knight Love

also α interferon in CHL
B in DS

antiviral oral

أهم علاج
في الـ DS

TREATMENT

لـ HCV مضيق

A. Treatment of HBV or HCV chronic active hepatitis:

ORAL (interferon) CAH

1. Alpha - Interferon:

- **Dose:** 10 million units in HBV, & 3 million units in HCV, SC, 3 times / week, for at least 6 months.
- **Side effects:**
 - **Body:** Fatigue.
 - **Brain:** Depression.
 - **BM:** Supression.
- **Results:** Response occurs in 30 % of patients.

روسته
يوم آه ويوم لا
وقت الحلد
مدة 6 شهور لسنة
سفي وابتاجه بصورة دم

OR
والاينس
 α -interferon
والفهرس
long & short
acting

2. Pegylated Interferon:

"Long acting Interferon"

- **Dose:** 180 ug, SC, once / week for at least 6 months.
- **Side effects:**
 - **Body:** Fatigue.
 - **Brain:** Depression.
 - **BM:** Supression.
- **Results:** Response occurs in 60 % of patients.

من علاج CAH بأخذ
interferon
A: if not passes in Cirrhosis
أما العلاج على تليف لـ cirrhosis transplant
لأنه أنت بتعطى فقط interferon عاير مع
أكن الحقيقة فيه مدرسة بتقول أنت
حتى لو فيه Cirrhosis
أحتاج و جاز
أدى 70%
له
interferon
عسان
أمنع أنه
يرجى
HCC

دى مشكلة الوحيدة
V.V.V.V. expensive
الحقنة الواحدة
1800 LE
in week
50 weeks
= 90 thousands LE

3. Other antiviral agents:

e.g.

- Ribavirin: is added to Alpha - Interferon for HCV.
- Lamivudine: is added to Alpha - Interferon for HBV.

⊕
Ribavirin
15 thousands

B. Treatment of autoimmune chronic active hepatitis:

= 0.5 or 1 mg/kg/day as usually their wt is 30-40-45 kg

- 1. **Prednisolone:** 30 mg / day for one week, then 15 mg / day for 6 months to 3 years.
- 2. **Azathioprine:** 50 mg / day added to prednisolone to potentiate its effect & reduce its dose.

plus ⊕

ORAL

فايد
Azathioprine
S.N.I.

C. Hepatic transplantation.

1 if cirrhosis
2 if not respond to α interferon 70%
Pegylated 40%
White Knight Love

HEPATIC TUMOURS

I. BENIGN TUMOURS:

e.g. Hepatocellular adenoma.

II. MALIGNANT TUMOURS:

- Primary: Hepato Cellular Carcinoma (HCC) = Hepatoma.
- Secondary: Metastases.

سؤال

HEPATOCELLULAR CARCINOMA

1 INCIDENCE

1. Age: usually over 40.
2. Sex: more in males (male: female is 3:1).
3. Predisposing factors:

- Hepatic cirrhosis: especially post-hepatic, hemochromatosis, alcoholic.
- Hepatitis viruses: C, B. (= virus → HCC) acute cirrhosis و هذه يفرقة
- Aflatoxin: a fungus metabolite that contaminates stored food. البجاجة/الخيمات
- Androgenic steroids: and also CCPs.

2 CLINICAL PICTURE

Symptoms

1. Asymptomatic: accidental discovery during ultrasonography or CT scan.
2. Pain: dull aching pain in the right hypochondrium.

Bronchogenic Co.

asympt.
Chest
metastasis
non

أسباب تشبه تشخيص cirrhosis قلب إلى HCC

ORAL

Criteria to suspect HCC in a patient with Liver cirrhosis

1. Rapid deterioration of his condition. Compensated (child A) → decompensated (child B)
2. Refractory ascites and encephalopathy. Child A بقى Child B
3. Right hypochondrial pain or appearance of a local swelling. man

Cirrhosis
لا يعمل دمج

كان هناك الزيادة من دمج
 due to 1 - sudden deterioration of liver fn
 2 - anorexia (as HCC is debilitating)
 ↓ K White Knight Love
 of encephalopathy

كان ينفذ بالأدوية ولوقت لا

2 Signs

General:

Low grade fever, cachexia, jaundice.

Abdominal:

Liver is **enlarged**, **hard**, **irregular**, **nodular**, **TENDER**.
Ascites may be present (with features of malignancy).

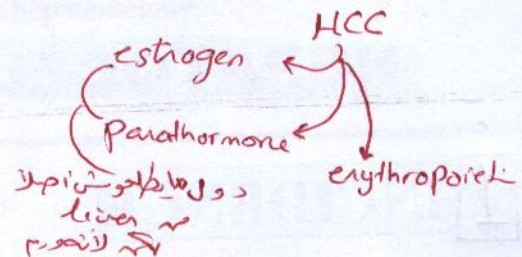
- 1 massive
- 2 Rapid accumulating
- 3 Hgic
- 4 malignant cells

3 Metastases

Brain, Bone, Lung, LN.

4 Non-metastatic presentations (Paramalignant syndrome)

e.g. Gynecomastia, Hypercalcemia.
estrogen
2ry polycythemia
(erythropoietin)
parathormone



3 INVESTIGATIONS

1. Liver imaging:

Abdominal ultrasonography, CT, MRI, Isotopic scanning.

2. Alpha Fetoprotein:

Refer to "Liver function tests".

3. Alkaline Phosphatase:

Markedly elevated.

liver → space occ. lesion
bone

3. Liver biopsy:

Better guided by ultrasonography or CT.

diffuse
cirrhosis

4 TREATMENT

operable

1. Surgical resection.

inoperable

2. Chemotherapy:

using Adriamycin.

3. Hepatic artery manipulation:

- **Embolization:** using gelatin foam via a catheter in the hepatic artery.
- **Ligation:** → HCC

4. Hepatic transplantation:

- In small unresectable tumours in patients with advanced cirrhosis.

Obsolete now as Recurrence occurs - new liver in about

White Knight Love

GRF malabsorption in Bacterial overgrowth?

- ① mucosal destruction & toxic effect
- ② splitting of Bile salts
- ③ Consuming of vit B₁₂

ازای زنگنه
absorption
TOP subject
oral و 1st و 2nd

MALABSORPTION

DEFINITION

- Defective absorption of any or all of the food components absorbed from the intestine.
- The main defect in absorption involves: FATS, leading to steatorrhea "fatty stools".

ETIOLOGY

I. IMPAIRED DIGESTION

1. Gastric causes:

- Gastric surgery: e.g. gastrectomy.
- Gastric carcinoma: decreased HCL → bacterial overgrowth in the intestine.
- Gastrinoma: increased HCL → inhibition of pancreatic lipase.
↳ Zollinger ellison syndrome

2. Pancreatic causes: (juices important for digestion)

- Chronic pancreatitis.
- Cystic fibrosis of the pancreas (mucoviscidosis).

II. REDUCED BILE SALTS

1. Hepatic causes:

- Acute & chronic hepatitis.

2. Biliary causes:

- Obstructive jaundice.

III. SMALL INTESTINAL DISEASES:

1. Amyloidosis.

infiltrates mucosa → obstruction of lacteal
فلا تعبره فـات بالذات

2. Bacterial overgrowth:

"Stagnant (blind) loop syndrome"

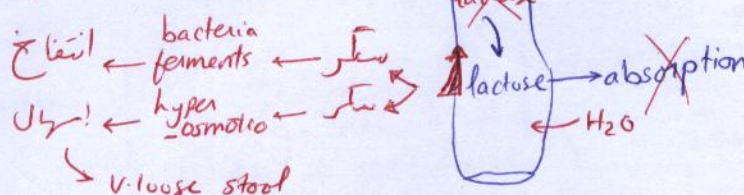
- Strictures Diverticulosis, Diminished motility (e.g. DM).

3. Coeliac disease:

"Gluten - sensitive enteropathy"

4. Disaccharidase deficiency: congenital (v. common in Egyptians)

- Lactase deficiency → malabsorption of lactose → bloating & diarrhea.



مرتب بین الاینها
لو مشربش
لا مکن

5. Ileal resection:

"short bowel syndrome"

- This will lead to interruption of the entero-hepatic circulation of bile.
- ↓ S.A. for absorption

6. Infections:

- TB.
- Tropical sprue.
- Whipple's disease.
- Parasites: e.g. Giardia Lamblia.

① mucosal destruction
② + TB → obstruct lactals

7. Irradiation enteritis.

8. Inflammation:

- Crohn's disease.

9. Lymphatic obstruction:

- Lymphoma.

U.C. bi
Colon
بس

Oral hazard of IRRadiation

Pericarditis → effusion

pleurisy → effusion

Non infective pneumonia

Aplastic anemia & leukemia

Irradiation enteritis

Venoocclusive disease

1 TB
2 lymphoma
3 Amyloidosis

IV. DRUG - INDUCED

- Cholestyramine (in hepatitis, in Biliary emboli)
- Neomycin (in Htt of Hepatic encephalopathy)
- Dendivan. (oral anticoagulant)

V. MISCELLANEOUS

- Heart: Congestive heart failure & Constrictive pericarditis.
- Thyroid: Myxoedema. → GRF

1. slow absorption ↓ thyroxine → metabolism
2. slow motility (→ Bacterial overgrowth) DM 5/

CLINICAL PICTURE

I. General features of malabsorption:

- Steatorrhea: stools are bulky, greasy, offensive, difficult to flush.
- Diarrhea. by 4 mechanisms (osmotic, secretory, motility, inflammatory)
- Abdominal distension, colics, borborygmi (audible intestinal sounds).

myxoedema (مخوادم), زيادة
الوزن و constipation
malabsorption

في زيت
fat

II. Specific features of malabsorption:

- Specific clinical features according to the deficiency of specific factors.
- See the following table:

Deficiency of specific factors

Fats:	Loss of weight. & (steatorrhea generally)
Proteins:	Muscle wasting & nutritional oedema. (DD of generalized edema)
Carbohydrates:	Hypoglycemia. (except pancreatic causes) نقص في سكر الدم (ما عدا أسباب البنكرياس)
Vitamin A:	Night blindness.
Vitamin D:	Rickets إطالة or osteomalacia. كسار
Vitamin E:	Dermatitis.
Vitamin K:	Bleeding tendency that is corrected by parenteral vitamin K.
Vitamin B1:	Beri - Beri.
Vitamin B2:	Glossitis & angular stomatitis. اللثة الوردية
Nicotinic acid:	Pellagra. فشل الكبد
Vitamin B6:	Peripheral neuropathy.
Vitamin B12:	Megaloblastic anemia & SCD. vit C: scurvy
Folic acid:	Megaloblastic anemia. → depends on which is dominant (megaloblastic)
Iron:	Microcytic anemia.
Sodium:	Muscle cramps & hypotension.
Potassium:	Myopathy & arrhythmias.
Calcium:	Parasthesia & tetany.
Magnesium:	Parasthesia & tetany.
Water:	Dehydration.

III. Features of the cause:

1. Coeliac disease "Gluten - sensitive enteropathy"

- Etiology:

Unknown (Autoimmune) Most common cause of Malabsorption 52%

- Unexplained sensitivity to "gluten" which is a protein present in wheat & barley.
- Unexplained mucosal damage by "gliadin" which is a fraction of "gluten".

- Clinical picture:

- Age: most commonly 20 - 40 years.
- Presentation: severe malabsorption syndrome + dermatitis herpetiformis.
- Associations: other autoimmune diseases, e.g. autoimmune thyroiditis & IDDM.
- Complications: increased incidence of lymphoma & small intestinal carcinoma.

- Investigations:

- Jejunum biopsy: Villous atrophy (**the most important investigation**).
- Serology: IgA antigliadin antibodies, anti endomysial Ab (IgA)
- General investigations for malabsorption.

Specific not sensitive
لؤلؤة أخلف

- Treatment:

- Withdrawal of all gluten from the diet.

Oral

- Biopsy: villous atrophy
- eliminate gluten from diet → improves (therapeutic test)

endomysium = C.T. surrounding smooth M.s of intestine

chronic skin disease characterized by persistent itching, lesion: vesicles & papules in g.p.s

2. Tropical sprue

- **Etiology:** Unknown (infection)
 - May be bacterial infection.
- **Clinical picture:**
 - Severe malabsorption, especially of: fats, folic acid & cobalamin (= B₁₂)
 - Associated mouth ulcers.
- **Investigations:**
 - Jejunal biopsy: Villous atrophy (by infection) (the most important investigation).
 - General investigations for malabsorption.
- **Treatment:**
 - Antibiotic: Tetracycline 250 mg / 6 hours orally daily.
 - Replacement: Folic acid & cobalamin.

3. Whipple's disease

- **Etiology:** Unknown (infection)
- **Clinical picture:**
 - Steatorrhea.
 - Fever, arthritis, lymphadenopathy, cranial nerve affection.
- **Investigations:**
 - Jejunal biopsy: heavy infiltration by macrophages.
 - General investigations for malabsorption.
- **Treatment:**
 - Antibiotic: Trimethoprim – sulfamethoxazole.

- tetracycline استخلاصات
- 1 Tropical sprue
 - 2 Cholera, malaria & Brucella
 - 3 pleurodisin
 - 4 pericarditis
 - 5 Atypical pneumonia
 - 6 Recurrent pneumothorax
- to avoid malignant effusion

INVESTIGATIONS

I. Investigations to diagnose malabsorption:

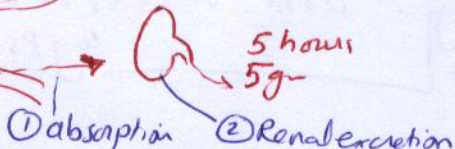
1. Fat in the stools:

- Normally: fat in the stools is not more than 7 gm / day.
- In steatorrhea: total fat is increased:
 - Non – split: in impaired digestion. stomach العيب
 - Split: in small intestinal diseases. العيب

2. Urinary D – Xylose test: Most imp

- Method: give 25 gm D – Xylose orally, then collect urine over the next 5 hours.
- Normally: urine collected over 5 hours should contain at least 5 gm of D – Xylose.
- In steatorrhea: urine collected over 5 hours will contain less than 5 gm of D – Xylose "provided that renal functions are normal".

absorption renal function يفتقر الحليم بس



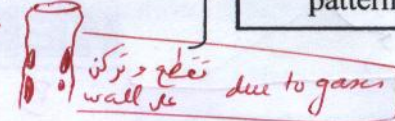
3. Barium follow through of the small intestine:

- Loss of the normal feathery appearance of the jejunum.
- Segmentation & flocculation of the barium.
- Dilatation of the intestinal lumen.

Malabsorption pattern

4. Jejunal Biopsy:

- Recently: taken through the enteroscope.
- Previously: taken by the intestinal biopsy capsule. *Obsolete*

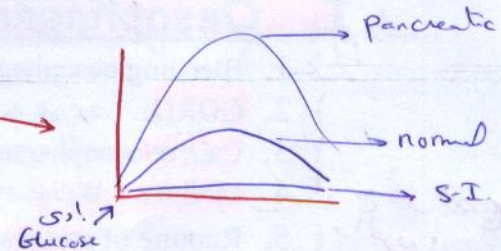


2 x ray
malabsorption
- U.C.
miscellaneous
chest, heart

II. Investigations to diagnose the cause:

1. Glucose tolerance test:

- In small intestinal diseases: flat curve.
- In pancreatic causes: diabetic curve.



2. Tests for Bacterial overgrowth:

♥ ¹⁴C - Xylose breath test:

- Method: measure the ¹⁴CO₂ in the breath after oral administration of ¹⁴C - Xylose.
- In bacterial overgrowth: ¹⁴CO₂ in the breath will increase because more bacteria will act on the ¹⁴C - Xylose.

♥ Culture of the jejunal aspirate:

- This is done using a sterile polyethylene tube.

III. Investigations for the deficient factors:

1. Blood picture:

microcytic or megaloblastic anemia.

2. Plasma proteins:

hypoproteinemia.

3. Serum electrolytes:

diminished Iron, Na, K, Ca, Mg.

4. Bone X - ray:

osteomalacia.

TREATMENT

1. Treatment of the cause:
2. Replacement of the deficient factors:

e.g. anti-TB drugs for TB.
e.g. parenteral vitamins & minerals.

Leg: vit K → bloody stool
Fe → deficiency

Oral Q:

ما هي الـ واحد يطرد دم أحمر
و رغم كونه عند hematemesis
أ: لو اختل شرطه الثلاثي دول

GIT BLEEDING

HEMATEMESIS

Blood vomit

HCl إما مفيش

(Achlorhydria) or (Antacids as H₂ Blockers)

وقت أقل نسبه
(massive bleeding)

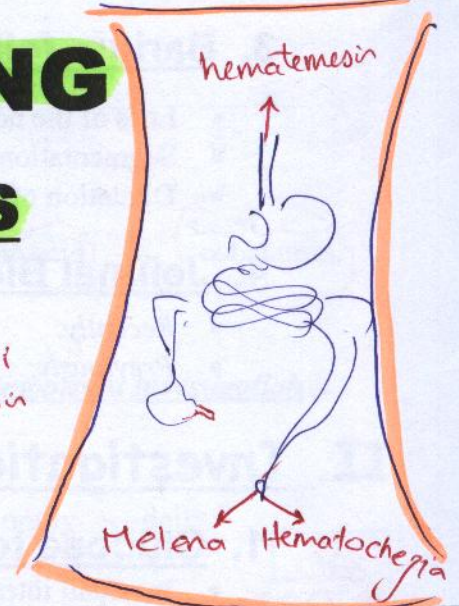
1 DEFINITION

- Vomiting of blood.

2 ETIOLOGY

بني غامو
① as HCl on Blood
② > 1 hour Contact
acid hematin (coffee ground)

إما لو أحمر
Hemoptysis



I. Oesophageal causes:

1. Bleeding oesophageal varices.
2. GORD: acid in wrong site causes oesophageal erosions above
3. Cancer oesophagus.
4. Mallory - Weiss syndrome: tear of the oesophago - gastric junction due to **severe vomiting**.
5. Rupture of aortic aneurysm into the oesophagus. in post mediastinum

مع كثر التقيح
والخزيمه الـ
oesophago gastric
junction
تتبرح

II. Gastro - duodenal causes:

1. Acute gastritis: superficial erosion due to drugs, especially aspirin, NSAIDs, corticosteroids.
2. Acute gastric ulcer: deep erosion due to "drugs", especially aspirin, NSAIDs, corticosteroids, OR due to severe stress "Stress ulcer".
3. Chronic peptic ulcer: gastric ulcer OR duodenal ulcer.
4. Bleeding gastric varices: fundal varices.
5. GORD: associated with hiatus hernia.
6. Cancer stomach: and cancer ampulla of Vater.
7. Peutz - Jeghers syndrome: GI polyposis & muco - cutaneous pigmentation.
8. Angiodysplasia of the stomach.

III. Upper small intestinal causes:

1. Angiodysplasia of the small intestine.
2. Familial polyposis: adenoma.
3. Cancer: adenocarcinoma.
4. Ulcers: secondary to drugs as NSAIDs OR typhoid ulcers.

؟؟

بعض ركب
كاتبها

حول الفم
buccal
mucosa

IV. General causes:

1. Hemorrhagic blood diseases: purpura, hemophilia & leukemia.
2. Hemorrhagic fevers: plague.
3. Hypertension: severe hypertension.

② - Peutz-jegher
① - Addison

V. False hematemesis:

- Vomiting of ingested blood coming from: bleeding nose, mouth OR pharynx.

epistaxis cerebral
Common serious

لأنهم
أما النزيف لرحله من الفم

3 DIFFERENTIAL DIAGNOSIS

- Differentiation from hemoptysis: refer to the chest.
- Differentiation from false hematemesis: by local examination.
- DD of the cause of hematemesis through:

1. Clinical picture.

2. Investigations:

barium meal
ایک میٹال

- Upper endoscopy
- Barium meal
- Liver function tests
- Angiography

stomach

& enteroscopy.

& follow through.

& abdominal ultrasonography.

& isotopic studies.

duodenum

[to deficiency anemia]
for angiodysplasia

varices

4 TREATMENT

- Treatment of the cause.

[P.U.
varices: injection sclerotherapy]

MELENA

1 DEFINITION

- Passage of black tarry stools due to the presence of digested blood.

2 ETIOLOGY

- Same causes of hematemesis.

ligament of Treitz (diaphragm to duodeno jejunal)

3 DIFFERENTIAL DIAGNOSIS

- DD of dark stools:
 1. Intake of iron.
 2. Hemolytic anemia. (jaundice الوبحة)
- DD of the cause of hematemesis.

stools dark
stercobilinogen

hematemesis
melena
fresh blood

وینفع حاجه رقوم هذا lig

4 TREATMENT

- Treatment of the cause.

hematemesis

نزل Fresh
Massive & rapid
digestion

BLEEDING PER RECTUM

ETIOLOGY

- Piles: the most common cause.
- Cancer rectum OR colon: the most serious cause.
- Inflammatory bowel disease: especially UC.
- Intestinal bilharziasis.
- **A**nal fissure.
- **B**acillary dysentery.
- **C**olonic polyps: familial polyposis.
- **D**iverticulitis.
- TB of the colon.

TREATMENT

- Treatment of the cause.

DIARRHOEA

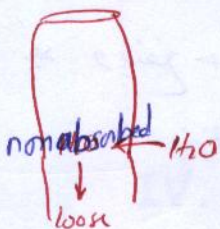
DEFINITION

- Passage of increased amounts of loose stools.

PATHOGENESIS

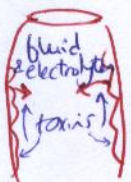
1. Osmotic diarrhea:

- Due to: presence of high concentration of non-absorbed hypertonic substances in intestine which will attract fluid from the blood to intestine → loose stools.
- Examples: In disaccharidase deficiency: high concentration of lactose.
In malabsorption syndrome: high concentration of solutes.
Ingestion of non-absorbable substances, e.g. lactulose.



2. Secretory diarrhoea:

- Due to: active intestinal secretions of fluid & electrolytes.
- Examples: Enterotoxins: cholera & E.coli.
Hormones: e.g. Vaso-active intestinal peptides "VIPs".



→ normally in intestine

→ VIPoma (pancreatic cholera)

White Knight Love

3. Inflammatory diarrhoea: " Mucosal destruction "

- Due to: 1 damage to the intestinal mucosa, causing loss of fluid & blood.
2 defective absorption.
- Examples: Bacillary dysentery.
Ulcerative colitis.

[only in cases of small intestine]

UC: 1 only
Crohn: 1, 2



DM Hyxedema

4. Abnormal motility:

- Due to: Hypermotility which causes defective absorption.
- Examples: Thyrotoxicosis & post-vagotomy.

/ also Hypomotility

↓ contact time c mucosa (intestinal hurry)

malabsorption

stagnation → Bacteria overgrowth

diarrhea malabsorption

CAUSES OF ACUTE DIARRHOEA

I. INFECTION

most common

inflammatory
secretory

A. Bacterial:

1. Shigella: Bacillary dysentery.
2. Cholera.
3. E.coli.
4. Food poisoning: salmonella, staph. & clostridium.

due to upregulation of serotonin Receptors.
more sensitive to action of serotonin

secretory

B. Viral:

1. Rota virus: watery diarrhoea & vomiting in children & infants.
2. Norwalk virus: diarrhea & vomiting in older children & adults.
3. Echo virus.

inflammatory

C. Protozoal:

1. Ent. Histolytica.
2. Giardia Lamblia.
3. Malignant malaria.
4. Balantidium coli.

Inflammatory & secretory

D. Helminthic:

1. Ascaris.
2. Ankylostoma.
3. Strongyloides stercoralis.

II. TOXIC:

- Arsenic, lead, some types of mushrooms.

III. DIETARY:

- Osmotic
 - Inflamm & secretory
 - motility
1. Excessive intake of cellulose.
 2. Food allergies.
 3. Unripe fruits, spices.

IV. NERVOUS:

- motility
- Exaggerated gastro - colic reflex: e.g. before examination.

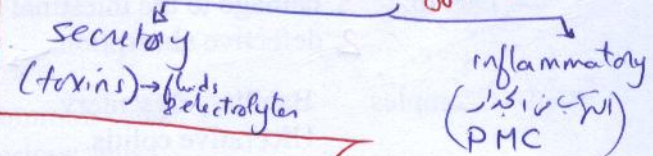
V. MISCELLANEOUS:

- inflammatory
1. Appendicitis.
 2. Ischemic colitis.

Oral : clindamycin → pseudo membranous colitis

harmful bacteria (cl. difficile) ← inhibits good bacteria flora

* these drugs inhibit ↑ so, harmful bacteria flare up cl. difficile



1. diffuse hyperemia & edema
 2. exudative plaques
 - (A) cells → RBCs, pus cells, necrotic mucosal cells
 - (B) fibrin
 3. skip areas in between
- membrane

CAUSES OF CHRONIC DIARRHOEA

A. Diseases of the colon:

- inflammatory
 - inflammatory
 - inflammatory
 - inflammatory
 - inflammatory
 - inflammatory
 - hypomotility
1. Amoebic colitis. *acute chronic*
 2. AIDS: due to opportunistic infections. *CMV*
 3. Bilharzial colitis. (polyps & ulcers)
 4. Cancer colon. (sporadic) (false)
 5. Crohn's disease of the colon OR ulcerative colitis. *I BD*
 6. Diverticulosis.

B. Diseases of the small intestine:

- Same causes of intestinal malabsorption. *کسری*

C. Endocrinal causes:

- hypo > hyper
 - inflammatory
 - hyper & hypo motility
 - secretory
 - hypermotility
1. Diabetic neuropathy. *تقر*
 2. Thyrotoxicosis. & Myxedema
 3. Addison's disease.
 4. Gastrinoma: "Zollinger - Ellison syndrome". ↓ pancreatic lipase
 5. Verner - Morrison syndrome: "Pancreatic cholera"
 - This is a pancreatic tumour which secretes VIPs causing secretory diarrhoea.
 6. Carcinoid syndrome.

D. Drugs:

- Oral SQ
- ① Antibiotics: especially Clindamycin which causes pseudomembranous colitis.
 2. Purgative abuse.
 - 3- drugs causing malabsorption: Neomycin, dandelion, cholestyramine

E. Miscellaneous:

- inflammatory
 - mucosal damage
1. Obstructive jaundice.
 2. Pellagra. *3Ds*
 3. Vitamin B12 deficiency. → atrophy

11
Ampicillin

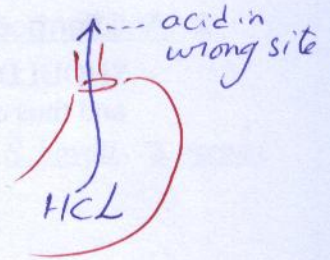
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cephalosporin

GORD

SQ

DEFINITION

- Clinical symptoms due to abnormal gastro – oesophageal acid reflux.



ETIOLOGY

1. Failure of anti-reflux mechanism:

due to failure of the tone of the LOS.

2. Dysmotility:

- Oesophagus:

poor oesophageal peristalsis leading to failure of acid clearance.

- Stomach:

delayed gastric emptying leading to increased chance for reflux.

3. Large hiatus hernia.

CLINICAL PICTURE

1. Heart burn: The major feature of GORD.

2. Pain: site Epigastric & retrosternal.
character Burning.

Increases on lying down.

Relieved by: antacids. & nitrates

DD. of Acute chest Angina
MI

3. Dysphagia:

& Regurgitation of food & acid into the mouth.

4. Hematemesis & melena:

due to mucosal inflammation and / or ulceration.

5. Chest Complications:

- Aspiration pneumonia.
- Nocturnal asthma (due to regurgitation & aspiration).

acid & food particles → Pharynx → if lost cough reflex
Flatulence → irritation → reflex spasm
URTIs & pneumonia

6. Other Complications:

- Barrett's oesophagus: mucosal metaplasia → ulceration or adenocarcinoma.
- Iron deficiency anemia: due to repeated bleeding.

DIFFERENTIAL DIAGNOSIS

- From other causes of acute chest pain especially angina & MI.

شرح
most imp criteria
of Acute chest Pain

INVESTIGATIONS

- GORD is a CLINICAL DIAGNOSIS & many patients can be treated without investigations.

1. Oesophagoscopy:

- It shows the presence of oesophagitis.

2. Barium swallow:

- It may show a hiatus hernia.

3. 24 – hour intraluminal pH monitoring: “The most important investigation”

- It shows the number of reflux episodes occurring over 24 hours.
- It is often combined with: manometry.

1. frequency
2. severe
3. Pressure

= marked
Changes –
peristalsis
(hypomotility)

تدخل قسطرة من مخرج
الى المريء وتثبت 24 ساعة
Transnasal

electrode
catheter

TREATMENT

A) Specific treatment:

1. Antacids: they form a gel with the gastric contents & thus reduce the reflux.
2. H₂ – receptor antagonists: e.g. Ranitidine.
3. Proton pump inhibitors: e.g. Omeprazole, “the drugs of choice”.
4. Prokinetic drugs: e.g. Metochlopramide increases the peristalsis.

histamine
++ HCl

ab
next
For 2M

B) General treatment:

1. Weight reduction.
2. Raising the head of the bed during sleeping.

C) Surgical:

Indication * failed Medical (2M) or 3M or even 6M

Operation < 1 for Hiatus Hernia
2 endoscopic suturing
3 Induce fibrosis at LOS by radiofrequency waves [Stretta]

also m. gramine

1000
ORAL

CAUSES OF ACUTE ABDOMEN

1. Acute appendicitis.
2. Acute cholecystitis.
3. Acute pancreatitis.
4. Acute intestinal obstruction.
5. Acute salpingitis OR twisted ovarian cyst.
6. Colic: biliary, renal, intestinal.
7. Perforated peptic ulcer.

Surgical
causes

بدرج غلط للجراح وفتحة

8. Acute myocardial infarction. ^{inferior}
9. Acute pleurisy & Pneumonia.
10. Diabetic ketoacidosis.
11. Uremia.
12. Hemolytic crisis. *Qualitative mesenteric ischemia*
13. FMF.
14. Food poisoning.
15. Rheumatic fever (non – infective peritonitis).

Medical
causes

Other criteria

GASTRITIS

Inflammation of the gastric mucosa; it may be: ACUTE or CHRONIC.

ACUTE GASTRITIS

Inflammation of the SUPERFICIAL gastric mucosa.

ETIOLOGY

1. DRUGS:

Aspirin, / NSAIDs. / cortisol

2. STRESS (Stress gastritis):

Sepsis, Shock, Severe burns (Curling's ulcer), stroke, MI

3. INFECTIONS:

CMV, HSV.

GRF ulcer in pt. stress

INVESTIGATIONS

Upper endoscopy confirms the diagnosis.

erosive gastritis
التهاب السطح

TREATMENT

1. Removal of the offending cause.

2. Drugs:

+ H₂ receptor antagonists (ranitidine 150 mg bid).
Proton pump inhibitors (omeprazole 20 mg once daily).

C/P ① dyspepsia & N&V
② Heart burn

③ Hematemesis & melena

investig. erosive gastritis

CHRONIC GASTRITIS

TYPES

1. Chronic active gastritis:

Helicobacter pylori.

2. Autoimmune gastritis:

Pernicious anemia.

هنا لا يتجيب لـ

also (applies by ca stomach)

3. Chemical gastritis: due to repeated chemical injury, e.g. bile reflux or chronic NSAIDs ingestion.

CLINICAL PICTURE

Most cases are asymptomatic: discovered accidentally at upper endoscopy.

symptomatic triad

Osteoarthritis

TREATMENT

Treatment of the cause: e.g. H. pylori.

nowadays regime

1- ✓

2- ✓

3. clarithromycin 250 mg 2/d

← classic regime

1w - 10d

3 drugs altogether (Triple therapy)

1 → Omeprazole 20 mg twice daily

2 → Amoxicillin 500 mg 4/d

3 → Metronidazole (even 6 hours) 500 mg 3/d

White Knight Love

INFLAMMATORY BOWEL DISEASE

DEFINITION

The term **IBD** refers to the: IDIOPATHIC chronic **I**nflammatory **B**owel **D**iseases including:

- ULCERATIVE COLITIS (UC).
- CROHN'S DISEASE (CD).

ETIOLOGY

UNKNOWN some theories were suggested:

1. **Immunological:**
2. **Iatrogenic:**
3. **Inheritance:**
4. **Ingested food:**
5. **Infection:**

presence of associated extra-intestinal autoimmune manifestations. *responds to immunosuppressive*
 prolonged use of NSAIDs. (Excluding Aspirin)
 genetic factors.
 milk, complex carbohydrates.
 by atypical mycobacteria (unproven theory).

INCIDENCE

- Age: young (20 – 30 years).
- Sex: more in females.
- Stress: is a precipitating factor.
- Smoking: protects against UC, **BUT:** is associated with CD.

ULCERATIVE COLITIS

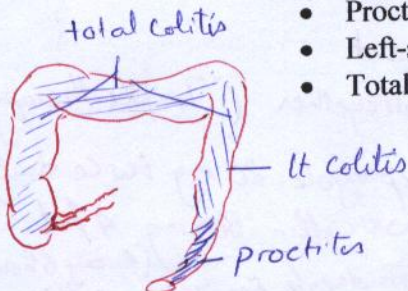
DEFINITION

A chronic inflammatory disorder of the mucosa of the colon.

PATHOLOGY

1. **Site:** "Diffuse, Continuous, Superficial inflammation of the **MUCOSA** only"

- Proctitis: affection of the rectum only.
- Left-sided colitis: affection of the rectum + distal colon.
- Total colitis: affection of the rectum + whole colon.



Small intestine skip area → Crohn's thickness ↑

Proximal spread

2. Lesion: "superficial affection"

- Multiple ulcerations.
- Multiple CRYPT ABSCESES: infiltration of the crypt lumen by excessive neutrophils.

CLINICAL PICTURE

I. INTESTINAL MANIFESTATIONS

A. Acute attack:

- Bleeding per rectum: ulcers is the most important feature.
- Diarrhoea with mucopus: may occur more than 10 times daily.
- Tenesmus: sense of incomplete evacuation due to colorectal irritation
- Lower abdominal pain: colicky.

B. Chronic disease:

- After recovery from the acute attack, the patient returns to a normal bowel habit.
- Then the disease runs a chronic intermittent course characterized by:
 - Acute exacerbations: precipitated by **NSAIDs** or **Nervous stress**.
 - Remission periods: which may be very short & thus need continuous ttt. (maintenance)

II. EXTRA - INTESTINAL MANIFESTATIONS

Extra - Intestinal Manifestations of UC

- General:** Fever ^{Inflammation} + general constitutional manifestations.
- Nutritional:** Anemia ^{GRF} + manifestations of vitamin deficiency + PLE. ^{malabsorption}
- Eye:** Conjunctivitis + iridocyclitis.
- Skin:** Erythema nodosum + pyoderma gangrenosum + vasculitis.
- Joints:** Arthritis. ^{Auto-immune}
- Liver:** Gall stones + autoimmune hepatitis, hepatomegaly.
- Kidney:** Oxalate stones.
- Lungs:** Interstitial pulmonary fibrosis.

Edge: Black gangrenous
margin: Erythematous
Surface: necrotic + blood
+ pus

ORAL

skin manifests of u.c.

ORAL

oral manifestations

oral manifestations

oral manifestations

oral manifestations

1 IBD

2 DM

3 Hyperparathyroidism

COMPLICATIONS

1. Hemorrhage.
2. Perforation.
3. Megacolon: acute (toxic) dilatation of the colon.
4. Malignancy: increased risk in cases of "total colitis of 10 years duration".

toxic mega colon

لذلك لا يفر
Proctocolectomy
= (total colectomy)

INVESTIGATIONS

1. Stool analysis & culture:

- To exclude an infective colitis.
- Presence of: WBCs, RBCs due to inflammation.

هذا ليس سبب العدوى
infection

2. COLONOSCOPY:

- Lesion: diffuse inflammation, multiple ulcerations, friable mucosa.
- Biopsy: multiple CRYPT ABSCESSSES.

no skip areas

لا تتركز الدم (مفرقة)

كل ما كانت أكثر
بقى more severe

3. Barium enema:

- Lead - pipe appearance: loss of haustrations + narrowing of the lumen.

①
sacculation

②
due to fibrosis

4. Blood tests:

- Anemia, Leucocytosis.
- Abnormal liver function tests.

if autoimmune hepatitis associated

TREATMENT

I. MEDICAL TREATMENT

A. ACUTE ATTACK:

1. Steroids:

"the most important ttt for the acute attack"

- Prednisone, orally 1 mg / Kg / day: in acute attacks.
- Hydrocortisone IV 100 mg / 6 hours: in severe attacks (for 5 days).
- Hydrocortisone retention enema: in severe proctitis.

Value ← anti-inflammatory
immunosuppressive

2. Salicylates:

- Sulphasalazine (sulpha + salicylates): 1 gm / 6 hours orally.
- Mesalamine (5-aminosalicylic acid): 1 gm / 6 hours orally.

جرعات

4 gm / d

3. Symptomatic ttt:

- Anti-diarrheal agents: for severe diarrhoea.
- Antibiotics: for severe secondary bacterial infection.
- Anti spasmotic: for colic

on top of inflammation

B. RESISTANT CASES:

- Immunosuppressives: Azathioprine & Cyclosporine may be beneficial.

C. MAINTENANCE:

- Sulphasalazine: 2 gm orally daily for 2 years.
- Mesalamine: 2 gm orally daily for 2 years.

$\frac{1}{2}$ dose.

II. SURGICAL TREATMENT → ORAL

Indications:

1. Failure of Medical tt.
2. Development of severe complications.
3. Total colitis for more than 10 years: because of high cancer risk.

Cancer على القولون

Operation:

The standard operation is: "Proctocolectomy with permanent ileostomy".

CROHN'S DISEASE

داء DD و SQ و malabsorption

أهم في الـ IBD
u.c r

DEFINITION

A chronic inflammatory disorder which affects:

- ① Any part of the GIT from the mouth to the anus.
- ② The whole thickness of the bowel wall: "Transmural".
- ③ Skip areas → حبيطة
- ④ multipockets, fissures, fistulae. & thickened & narrowed Malabsorption

القرون

u.c.

- ① - Colon only
- ② - Mucosa only
- ③ - No skip
- ④ - No malabsorption

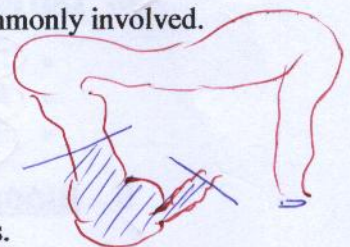
PATHOLOGY

1. Site: Any site but

- The TERMINAL ILEUM and RIGHT COLON are most commonly involved.

2. Lesion:

- Thickening & narrowing of the affected bowel.
- Ulcers, fissures, fistulas, abscesses.
- SKIP LESIONS: areas of normal bowel inbetween the lesions.



كانوا يسمونه
زنا
distal ileitis

CLINICAL PICTURE

I. INTESTINAL MANIFESTATIONS

- Acute colicky pain & tenderness: in the right iliac fossa (simulating acute appendicitis).
- Mass: (proliferative granuloma) in the right iliac fossa.
- Malabsorption syndrome: diarrhoea, steatorrhoea, loss of weight.
- Intestinal obstruction.
- Colonic CD: features similar to UC.

II. EXTRA - INTESTINAL MANIFESTATIONS

- Same as Ulcerative Colitis.

اشترط
لامنه

COMPLICATIONS

Same as Ulcerative Colitis, BUT:

- Malignancy: is less common in CD.
- Fissures, Fistulas, Abscesses: are more common in CD.

INVESTIGATIONS

1. Stool analysis & culture:

- To exclude an infective colitis.
- Presence of: WBCs, RBCs due to inflammation.

2. COLONOSCOPY & ILEOSCOPY:

- Lesion: diagnoses the characteristic pathology & detects colonic affection.
- Biopsy: differentiates between CD and UC.

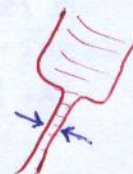
3. Barium enema:

- To detect colonic affection.

4. Barium follow-through:

- Deep ulcerations.
- String sign: areas of narrowing.
- SKIP LESIONS: areas of normal bowel inbetween the lesions.

string sign
of Kantor



5. Blood tests:

- Anemia, Leucocytosis.
- Abnormal liver function tests.

المظهر
و أكثر
من الشدة

TREATMENT

I. MEDICAL TREATMENT

تبرأ Prednisone: لوما نفخش تصيف Azathioprine

A. ACTIVE ILEITIS:

- Prednisone: orally 1 mg / Kg / day.
 - Immunosuppressives: Azathioprine & Cyclosporine may be beneficial in resistant cases.
- تصيف لوما نفخش

B. ACTIVE COLITIS:

تبرأ Prednisone & sulpha: لوما نفخش تصيف Azathioprine

- Prednisone: orally 1 mg / Kg / day.
 - Immunosuppressives: Azathioprine & Cyclosporine may be beneficial in resistant cases.
 - Sulphasalazine (sulpha + salicylates): 1 gm / 6 hours orally.
- تصيف لوما نفخش

C. ANAL DISEASE:

- Metronidazole: 800 mg tds orally.

Metronidazole

- P.U. (Helicobacter)
- Anaerobes
- Neomycin
- Antiamoebic
- Anti giardia

II. SURGICAL TREATMENT

Indications:

1. Failure of Medical ttt.
2. Development of severe complications.
3. Failure of growth in children. → due to malabsorption

Operation:

- Standard: Limited resection with end to end anastomosis.
 - In severe colitis: Proctocolectomy with permanent ileostomy.
- terminal

INTESTINAL TUBERCULOSIS

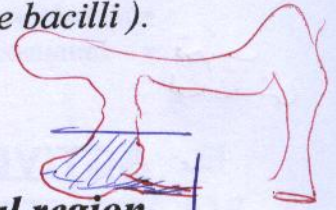
ETIOLOGY

- **Secondary:** to pulmonary TB (Human bacilli).
- **Primary:** ingestion of infected milk (Bovine bacilli).

Blood spread

PATHOLOGY

- The most common affected site is the ileo-cecal region.



CLINICAL PICTURE

- General Symptoms: night fever, night sweating, loss of appetite, loss of weight.
- Abdominal Symptoms: abdominal pain, diarrhoea, **MALABSORPTION SYNDROME**.
- Signs: abdominal tenderness, mass in the right iliac fossa.

دفع و TB من الأمعاء إلى الدم

NB Evidence of Pulmonary TB may be present.

apical Cavitation or fibrosis

Rt iliac fossa

① inflammatory

② obstructed lacteals

COMPLICATIONS

- Bleeding.
- Perforation.
- Fistula.
- Obstruction. (man)
- General complications of TB (refer to Chest).

السرطان

- Aspergill
- Angiodoz
- spread
- of drug

DIFFERENTIAL DIAGNOSIS

1. Crohn's disease. inflammatory granuloma malabsorption Per: & terminal } both.
2. Causes of Malabsorption syndrome.

حزب 1
CLP investigation

malabsorption
due to TB

INVESTIGATIONS

1. Enteroscopy or colonoscopy and biopsy: for pathological diagnosis.
2. Sputum culture for TB + CXR

TREATMENT

- Anti- Tuberculous drugs.

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Bacillary
Intest. Annelid } *1 pie*
2001

IRRITABLE BOWEL SYNDROME

19

DEFINITION

A group of abdominal symptoms for which no organic cause can be found.

ETIOLOGY

1. Motility disturbances: increased or decreased strength of colonic motility.
2. Psychological disturbances: evidence of associated stress, depression, or anxiety.

CLINICAL TYPES

1. Spastic colitis: chronic abdominal pain & constipation.
2. Diarrhoea: chronic intermittent watery diarrhoea.
3. Alternating type: alternating constipation & diarrhoea.

CLINICAL PICTURE

1. PAIN:

- Character & site: chronic crampy pain in any part of the colon, esp. the left iliac fossa.
- Precipitating & relieving factors: pain ↑ following meals or stress & ↓ by passage of flatus or stools.

2. TENDERNESS:

- Over the left iliac fossa: and the spastic sigmoid colon may be palpable.

3. IRRITABLE MOTIONS:

- Chronic constipation, or diarrhoea, or both.
- **Ribbon-like stools** with sense of incomplete evacuation (Tenesmus).

4. IRRITABLE PERSONALITY:

- Features of anxiety or stress may be present.

DIAGNOSIS

Diagnosis of IBS is essentially a CLINICAL DIAGNOSIS.

Investigations are done to exclude organic causes:

- Stool analysis & culture.
- Sigmoidoscopy & rectal biopsy: *in cases of persistent diarrhoea.*

TREATMENT

1. Psychotherapy: reassurance & tranquilizers.
2. Symptomatic: High-fibre diet & laxatives for constipation, loperamide for diarrhoea, antispasmodics for pain.

MCQs

1. Bilirubin is:

- a) Derived exclusively from the breakdown of hemoglobin. *also myoglobin* X
- b) Bound in the unconjugated form to plasma beta-globulin. *Albumin* X
- c) Conjugated in the microsomes of the hepatocytes. ✓
- d) Reabsorbed in the small bowel as bilirubin diglucuronide. *stercobilinogen* X
- e) Normally excreted as stercobilinogen in the faeces & as urobilinogen in the urine. ✓

2. Hepatic encephalopathy in cirrhosis is typically precipitated by:

- a) Infection. ✓
- b) Hypokalemia. *& alkalosis* ✓
- c) Abdominal surgery. ✓
- d) Gastrointestinal bleeding. ✓
- e) Lactulose therapy. *it's Ht* X

3. The following features suggest extrahepatic cholestasis rather than viral hepatitis:

- a) A palpable GB. ✓
- b) Right hypochondrial tenderness. *وجود في اليمين* X
- c) Serum ALP conc. > 2.5 times normal. *marked* ✓
- d) Pruritus & rigors. *especially pruritis* ✓
- e) Peripheral blood polymorph leukocytosis. *viral = leukopenia & relative lymphocytosis* ✓

4. Typical liver function values in acute liver failure include:

- a) Hypoalbuminemia. *only - cholest* X
- b) Hypoglycemia. ✓
- c) Prolonged PT. *& not corrected by parenteral vit k* ✓
- d) Serum ALP > 3 times normal. *only moderately elevated* X
- e) Peripheral blood lymphocytosis. *is the cause* X

Sherif EL Hawary, MD

Sherif EL Hawary, MD

10. In refractory ascites due to liver cirrhosis:

- a) SBP is treated by cloxacillin. *SBP is cause of refractory ascites but treated by Ciprofloxacin* X
- b) Increased incidence of encephalopathy is a problem with TIPS. *shunting* ✓
- c) It may occur due to under or over restriction of salt. ✓
- d) Increased CVP may occur with Le Veen shunt. *hyper volaemia → Acute pulmonary edema* ✓
- e) Ascites is considered refractory when there is severe respiratory distress. X

(kuse)

11. Chronic liver cell failure:

- a) Palmar erythema is the most common skin manifestation. *Spider naevi* X
- b) Rapid increase in ascites occurs if varices rupture. *hge is a cause of Acute deterioration of ascites* ✓
- c) Each of pallor & cyanosis may be a clinical presentation. *to b to b to b* ✓
- d) It may lead to heart failure & renal failure. *↑ COP HRS* ✓
- e) Secondary diabetes is a common complication. *insulin resistance* X

only IGT not DM

12. In liver cirrhosis:

- a) ALKMA may be positive. *lupoid* ✓
- b) Anti - mitochondrial antibodies may be positive. *PBC* ✓
- c) Coomb's test may be positive. *lupoid is associated any autoimmune or PBC* ✓
- d) Anti - Schistosoma antibodies may be positive. *(if mixed pathology)* ✓
- e) Anti - nuclear antibodies & Anti - smooth muscle antibodies may be positive. *lupoid* ✓

13. Obstructive jaundice:

- a) Intermittent course suggests a gall stone or Gilbert's disease. *دوسو* X
- b) Cholestyramine relieves itching but may cause malabsorption. ✓
- c) Bleeding is always corrected by parenteral vitamin K. *also neonatal death* X
- d) It may complicate hemolytic & hepatocellular jaundice. ✓
- e) Xanthomata due to hypercholesterolemia are seen in the face. *Cholestatic viral hepatitis* ✓

14. Iatrogenic liver disease:

- a) Amiodarone causes cirrhosis. ✓
- b) CCPs cause cirrhosis. *Budd chiaci → Cirrhosis* ✓
- c) Paracetamol induces a dose - dependent liver toxicity. *cholestatic fatty liver* ✓
- d) Alpha methyl dopa causes hemolytic & hepatocellular jaundice. ✓
- e) Repeated blood transfusion leads to cirrhosis. *CAH* ✓

Sherif EL Hawary, MD

Hemochromatosis

TRUE OR FALSE

- Liver cirrhosis is effectively treated by anti-fibrotic drugs. X
- Liver transplantation is the only hope for terminal cirrhosis. ✓
- Alcohol may cause hepatitis, cirrhosis, fatty liver or obstructive jaundice. X
- In PBC, high serum IgM level is the most important investigation. Biopsy (Septal injury) X
- Corticosteroids are contraindicated in PBC. osteoporosis, anti-diabetic X
- Drugs causing biliary cirrhosis include: CCPs & Chlorpropamide. chlorpropamide X
- Budd-Chiari syndrome may present acutely by acute abdominal pain & ascites. irregular anti-neoplastic X
- Malignancy may be a cause of Budd-Chiari syndrome. invasion 1 - drug-induced H.A. 2 - aplastic
- A patient with malignancy is subjected to veno-occlusive disease. ✓ radio chemotherapy
- Liver cirrhosis may be caused by therapy with methotrexate. ✓
- In primary hemochromatosis, serum ferritin is increased. ✓
- In secondary hemochromatosis, serum ferritin is increased. ✓
- Cardiomyopathy is a reported manifestation of secondary hemochromatosis. (DCM, RCM) ✓
- In Wilson's disease, serum copper is increased due to lack of ceruloplasmin. هو زيادة في النحاس و النقص في السيرولوبلازمين X
- The most important complications of cirrhosis are: LF, PH & malignancy. ✓
- Viruses causing liver cirrhosis include: EBV, CMV, HSV & adenovirus. yellow X
- INH & Streptomycin are drugs reported to cause liver cirrhosis. X
- In the overflow theory of ascites, salt retention is primary & it starts ascites. 99% respiratory 1% GIT
- PH alone is a very important cause of ascites. X
- Hypoalbuminemia below 3.5 gm % will lead to ascites. (3) X
- Palmar erythema is the most common skin manifestation of liver cirrhosis. spider X
- In liver cirrhosis, thrombocytopathy & not thrombocytopenia will cause bleeding. X
- ↓ aromatic aa is responsible for the development of hepatic encephalopathy. ✓
- Tapping of ascites is contraindicated in severe liver cell failure. therapeutic ✓
- In liver failure, spironolactone is preferred to furosemide. ✓

- Severe hypoglycemia occurs in acute & not in chronic liver cell failure. ✓
- Hepatorenal synd is fatal when it occurs in an already compromised kidney. *begin* X
- In SBP, gm -ve organisms & Streptococci are responsible. *up to normal* ✓
- In SBP, ttt should be started with cephalosporins or ciprofloxacin. ✓
- Hypoalbuminemia is a marker of chronic liver disease. ✓
- Marked elevation of ALP is a marker of biliary obstruction. ✓
- Oral lactulose is the most important line of ttt of hepatic encephalopathy. *enema* X
- Urobilinogen in urine is increased in hepatocellular jaundice. ✓
- In hemolytic jaundice the color of urine is dark. *normal* X
- Serum transaminases are markedly elevated in CAH. *only acute* X
- AST is more specific for liver diseases than ALT. *Seri* X
- ALP is moderately elevated in hepatitis. ✓
- Gamma globulins are elevated in acute & chronic liver diseases. *antigenemia* ✓
- PT should be done before any invasive technique. ✓
- Alpha feto protein is markedly elevated in pancreatitis. *moderately* X
- In acute liver failure encephalopathy may develop within few hours. ✓
- Aspirin sensitivity in children may be a cause for acute liver failure. *Reye's* ✓
- Paracetamol may be a cause for acute liver failure. *not chronic* ✓
- Fatty liver may be complicated by acute fulmination. *if Acute Fatty - pregnancy* ✓
- In acute liver failure, liver enzymes are always elevated. → *Neuro* X
- Bilharzial hepatic fibrosis is the most common cause of ascites. → *liver* X
- Myxoedema is a reported cause for exudative ascites. → X *hypalbuminemia*
- Pericardial effusion causes ascites that follows oedema of both LLs. *transudative* X
- Malabsorption syndrome is a cause of ascites. *hypalbuminemia* *precex* X
- SBP auses rapidly deteriorating ascites or refractory ascites. ✓
- Severe ascites is associated with congested non-pulsating neck veins. X
- Refractory ascites may be caused by Hyponatremia or Hypernatremia. *only s/v* *per* *non phary* ✓

Sherif EL Hawary, MD

- diarrhea lactulose, add sugar
- Constipation X
- In TB peritonitis, diarrhea is a common manifestaion. ✓
 - Palpable abdominal masses is a common sign in TB peritonitis. ^{LNS} _{omentum} ✓
 - Cholesterol level is elevated in Obstructive jaundice. ^{normal} _{erythrocytosis} ✓
 - Pericarditis is a common complication of amoebic liver abscess. ^{not} _{not} ^{not} _{not} X
 - Amoebic liver abscess is a cause of fever with rigors. ^{not} _{not} ^{not} _{not} ✓
 - Amoebic antibodies are present in all patients with ameobic liver abscess. ^{90% only} X
 - Fatty liver may be caused by severe malnutrition. ^{NASH} ✓
 - In VH, chronic complications occur in HCV, HBV & HDV. ✓
 - Post-hepatitis syndrome is a rather benign complication of VH. ^{only psychol} ✓
 - GN complicates all types of VH. ^{only B, C, D} X
 - In PAN, hepatitis markers for B & C should be done. ✓
 - Hypoalbuminemia is reported in acute VH. X
 - HbeAg is associated with the lowest infectivity of HBV. ^{Ab, e, anti-HBe} _{Ag, bi, high} X
 - Cholestatic hepatitis is most common with HCV. ^A X
 - Acute hepatitis is usually severe in HCV infection. ^{mild} X
 - Piecemeal necrosis is a pathological feature of advanced CAH. ^{not} _{not} ^{not} _{not} X
 - Protein losing enteropathy may occur in Bilharziasis but not in crohn's disease. ^{not} _{not} X
 - Gaucher's disease is associated with huge hepatomegaly. ^{spleen} X ^{both}
 - UC & crohn's disease are known causes of malabsorption syndrome. ^{not} _{not} X
 - In Whipple's disease, jejunal biopsy reveals villous atrophy. ^{not} _{not} X
 - In malabsorption, a diabetic OGTT occurs in small intestinal causes. ^{not} _{not} X
 - Clindamycin is an important cause of antibiotic associated diarrhea. ^{not} _{not} X
 - Cancer colon may cause chronic diarrhea. ^{SPUR} ✓
 - Pancreatic cholera is a cause of osmotic diarrhea. ^{secretory} X
 - In intestinal ameobiasis, there is marked fever & toxemia. ^{only: badly} X
 - Tenesmus is a manifestation of dysentery. ✓
 - Tenesmus is a manifestaion of anal disease. ^{Colorectal} X

- 7
- In bacillary dysentery, there are flask-shaped ulcers with healthy mucosa in between. X
 - In bacillary dysentery, symptomatic ttt includes antispasmodics & antidiarrheals. X
 - Iron deficiency anemia is a reported complication in GORD. ✓
 - H2-receptor antagonist is the drug of choice for GORD. X
 - DKA represents a medical cause of acute abdomen. ✓ PPI
 - H pylori is a recognised cause for acute gastritis. chronic X
 - Pyoderma gangrenosum occurs in CD but not in UC. both X
 - Pyoderma gangrenosum occurs in association with gluten-sensitive enteropathy. X
 - Crypt abscess is the most important pathologic feature of CD. UC X
 - Malignancy occurs commonly in CD & UC. UC X
 - Skip lesions are characteristic of CD. ✓ 10 years epan cols X
 - Metronidazole is effective in UC. anal CD X
 - CD is a very important DD for intestinal TB. ✓ 2 HE 2 Int amoebiasis 1 abscess X
 - The jejunum is the most commonly affected site in intestinal TB. ileocaecal X
 - Ribbon-like stools occur characteristically in UC. IBS syndrome X

CASE 2

A female patient 38 years old with a long history of CCPs presented to the hospital with confusion, apathy, irritability, jaundice and flapping tremors. She had been completely normal one week ago.

1. What is your possible diagnosis??
2. What are the different causes of this fatal condition??
3. What are the causes of death in this condition??
4. How would you find the following in the investigations:

- o Plasma glucose.
- o Serum albumin.
- o Serum transaminases.
- o INR.

Sherif EL Hawary, MD

CASE 1

A 15 years old male patient presented to the hospital because he felt unwell and feverish since one week.

On examination:

- o Liver: shrunken liver, with sharp edge. stop
- o Spleen: enlarged. → splenomegaly

1 cirrhosis
2 β

Initial Investigations revealed:

- o Serum bilirubin: 1.5 mg / dL.
- o AST, ALT: moderately elevated.
- o CBC: anemia with reticulocytosis. anemia

H.A. $\rightarrow$ splenomegaly $\rightarrow$ β $\rightarrow$ cirrhosis

1. What are the 2 most likely possibilities?? Wilson
2. How can you explain the fever?? Pyrogens ② septicemia ③ Hemolysis
3. How would you investigate this patient to reach a proper diagnosis??
4. Enumerate the causes of shrunken liver.

15 y
early onset
= hereditary

General of α , anti
of wilson
- Cu $\downarrow$ serum
- Ceruloplasmin: $\downarrow$
- Cu $\uparrow$ urine
- Biopsy

CASE 2

A female patient 38 years old with a long history of CCPs presented to the hospital with confusion, apathy, irritability, jaundice and flapping tremors. She had been completely normal one week ago.

= Hepatic encephalopathy

1. What is your possible diagnosis??
2. What are the different causes of this fatal condition??
3. What are the causes of death in this condition??
4. How would you find the following in the investigations:

- o Plasma glucose. $\downarrow$
- o Serum albumin. $\oplus$
- o Serum transaminases. $\uparrow$ then $\downarrow$
- o INR. $\rightarrow$ prolonged

= Acute fulminant liver failure
= Acute Budd-Chiari Syndrome

Causes of fulminant

[]

CASE 3

A female patient 40 years old presented to the hospital with increasing fatigue and general ill health during the last year. She also complained of dull aching pain in her right hypochondrium since 6 months. The patient gave no history of previous blood transfusion or exposure to contaminated syringes.

On examination:

- o Eyes: no jaundice.
- o Abdomen: hepatosplenomegaly.
- o There were no manifestations of liver cell failure.

Initial Investigations revealed:

- o Serum bilirubin: 2 mg / dL.
- o AST: moderately elevated.
- o ALT: moderately elevated.
- o ALP: moderately elevated.
- o Serum albumin: 2.8 gm / dL.

1. What is your initial DD ??
2. Why couldn't you detect jaundice in the eyes of this patient ??
3. What is the type of the hyperbilirubinemia in this patient ??
4. How do you expect to find the urobilinogen in urine ??
5. Which of the abovementioned enzyme is most reliable in this setting ?? Why ??
6. What causes marked elevation of the abovementioned enzymes ??
7. Name 2 most important investigations that could help you detect the disease.

The patient was admitted to hospital for management.

Further Investigations revealed:

- o CBC: anemia.
- o Abdominal ultrasonography: splenomegaly + bright hepatomegaly.

1. What are the 2 most important investigations you would ask at this point ??
2. What is your overall DD at this point ??
3. What are the possible causes of the patient's anemia ??
4. What are the causes of bright hepatomegaly on ultrasonography ??
5. Describe the pathology of the patient's liver in this case.
6. How do you expect to find the PT in this patient ??
7. How would you treat this patient ??

Sherif EL Hawary, MD

Extrahepatic (Aplastic anemia)

ASSOC. Pericardial effusion
Ht: interloper
Fe, FA, B₂
Chronic hepatitis
White Knight Love
HA

① ACD (Chronic inflammation)
② Hypersplenism
③ Auto immune
④ Wilson
⑤ Bleeding (PTT) not corrected by FFP
⑥ Chronic hepatitis
⑦ ASSOC. Pericardial effusion
⑧ Extrahepatic (Aplastic anemia)

Chronic Active hepatitis
U/S Biopsy
Viral: B, C, D
Auto immune
Anti: Anti-ALKMA
Chronic HbsAg

ALT only liver
ALT Acute
AST Acute
ALP > 30 < 100, spleen enlargement, inflammatory Bone

Causes of CHA
Biliary Chronic
of 2/2

Chronic liver disease

subclinical (< 2.5)
chronic (Acute = normal albumin)
enzymes (AST, ALT)
stop 2.8 gm / dL

chronic hepatitis
Compensated liver
no CLP
silent
Hepatocellular
< 2.5 not high enough

U/S Biopsy
Chronic dis
Cirrhosis

Chronic Active hepatitis

① ACD (Chronic inflammation)
② Hypersplenism
③ Auto immune
④ Wilson
⑤ Bleeding (PTT) not corrected by FFP
⑥ Chronic hepatitis
⑦ ASSOC. Pericardial effusion
⑧ Extrahepatic (Aplastic anemia)

The patient was given her treatment & discharged. She was advised follow up. She came back one year later with generalized oedema.

1. Give 3 possible explanations for this oedema & discuss how to differentiate.
2. Mention the possible complications that this patient may develop.

CASE 4

A male patient 46 years old with a past history of **Bilharziasis** had been losing weight during the last year together with increasing manifestations of weakness and general ill health. He presented to the hospital with **jaundice and dark coloured urine** dating since 4 months. He said that he consulted a GP in his village who prescribed him some medications.

On examination:

- o Eyes: jaundice.
- o Uls: clubbing.
- o LLs: oedema of both LLs.
- o Skin: palmar erythema & echymotic skin patches.
- o Trunk: gynecomastia.
- o Abdomen: shrunken liver, splenomegaly & ascites.

1. What is your possible diagnosis ?? **LCF**
2. How would you investigate this patient to reach your proper diagnosis ??
3. What is the pathological role played by Bilharziasis in this patient ?? **PPF only**
4. What is the clinical stage of this disease ?? **stage 4 (shrunken liver, spleen, ascites)**
5. What is the most common skin manifestation in this condition ?? **SPIDER**
6. Explain the echymotic skin patches. How would you treat them ?? **bleeding tendency**
7. Discuss the pathogenesis of ascites in this patient.
8. What do you expect to find on examination of the patient's mouth ?? **PPF platelets**
9. Mention how can the odour of the mouth help you in diagnosis of diseases ??
10. Mention 2 possible causes of gynecomastia in this patient.
11. Explain the occurrence of clubbing in this case.

Sherif EL Hawary, MD

- Testosterone not destroyed
- the spermatogenesis

- pallor/cyanosis
- Fetus Hepatic
- vit Def. (angular / glazed)

WhiteKnightLove

The patient was admitted to hospital for management during which he developed acute fever, abdominal pain followed by deterioration in his consciousness level.

On examination:

- o Generalized abdominal tenderness.
- o Flapping tremors.

SBP

HE

1. What is your possible diagnosis ??
2. How would you confirm your diagnosis ??
3. How would you treat this condition ??

HE : clinically by flapping tremors
SBP : Ascitic fluid (3 cc) : polymorphs > 250/cmm

SBP : Apra & Cephalo
HE 2nd

CASE 5

A female patient 44 years old presented with a 5 months history of diarrhea associated with loss of weight & frequent abdominal pain. She also suffered from night sweats, night fever, numbness & tingling in her hands & feet.

3.5% : Chronic diarrhea + loss wt = Malabs. malabs.

On examination:

Pallor.

Pulse: 100 / min, with multiple extrasystoles.

stop Tenderness & mass in the right iliac fossa.

digestive disorders
hypokalemia

TB

Crohn's

malabs. + pain Rt. iliac fossa

1. What is your clinical diagnosis ?? malabs. \$
2. What are the 2 most likely etiological diagnoses arranged in order ?? 1 TB 2 CD
3. Mention one point you would like to examine in the patient. → chest
4. How can you differentiate between them by one investigation ?? → Colonoscopy & Biopsy
5. What are the complications of the most probable etiology ?? TB
6. Explain the numbness & tingling in this patient.
7. What could cause pallor to this patient ?? malabs. ↓ Fe, f, B₁₂
8. What are the laboratory tests you would like to request ?? TB & CD
9. What are the drugs that can cause chronic diarrhea ??

night sweats - 2 LBS

colonoscopy & Biopsy (TB granules)

frstulae - bloody - perforate - TB

general chest

11

Sherif EL Hawary, MD

- Antibiotic
- purgatives
- malabsorption drugs.

- 1 Malabs. 1 any D xylene
- 2 of Defecory
- 3 of T.B. : Bacteriology

WhiteKnightLove

Miscellaneous questions (enumerations)

1 - Discuss the importance of corticosteroids in liver & GIT diseases.

2 - Enumerate the causes of mixed jaundice.

3 - What are the markers of chronicity in HBV ??

HbsAg > 6M
HbcAg > 6M
HbeAg > 3M

Clinical – chemical diagnosis in hepatobiliary diseases (Liver screening)

- What is the most important laboratory parameter of hepatocellular integrity ??

Transaminases (ALT, AST)
 ALT > 40 U/L
 AST > 40 U/L

- What is the most important laboratory parameter of biliary integrity ??

ALP, GGT, SNT
 ALP > 120 U/L
 GGT > 60 U/L
 SNT > 100 U/L

- What is the most important laboratory parameter of hepatocellular synthesis ??

Albumin, PT, PC, INR
 Albumin < 3.5 g/dL
 PT > 14 sec
 PC > 1.2 sec
 INR > 1.5

- How would you screen cirrhotic patients for early detection of HCC ??

① AFP (liver)

- What are the important tumour markers in GIT diseases ??

AFP
 U/S
 Carbohydrate Ag
 ② Pancreas (CA 19-9)
 ③ Colorectal (CEA)

- What are the important autoantibodies in GIT diseases ??

① Hereditary (Dup. Johnson & rotor)
 ② Drug. (CCPs, chlorpromazine)
 ③ Pregnancy (estrogen)
 ④ Viral hepatitis (decreased)

Anti Mito
 Lipoid
 Anti nuclear
 Anti SM-Ms
 ALKMA
 Gluten
 Anti gland
 anti endogly

- Enumerate causes of nonobstructive cholestasis.

- Enumerate the causes of chronic diarrhea with abnormal findings on endoscopy.

- Enumerate the causes of chronic diarrhea with normal findings on endoscopy.

- ⑦
1. USES
 - ① Chronic ^{active} Hepatitis : lupoid (azathioprine)
 - Acute " : if cholestatic (# edema)
 - ② IBD
 - ③ TB peritonitis (under cover Anti-TB)
 - + ④ If liver transplant (Immuno suppressant)
 2. Side effects :
 1. fatty liver
 2. Gastritis & P.U. up to severe GIT Bleeding

- ⑧ Mixed jaundice : (HC)
1. viral Hepatitis → OJ (HAV)
 2. Haemolytic → OJ
 3. Biliary cirrhosis (OJ → H.C)
 4. Hypersplenism (H.C → H.J)
 5. CCPs → (OJ & H.C)
 6. α-methyl dopa → H.C & H.J
 7. Wilson → H.C & H.J

* Chronic Diarrhoea e abnormal imaging

⇒ Large int
 Colon : amoebic colitis : flask ulcers
 \$: polyps, ulcers

Cancer colon
 Crohn
 U.C.
 Diverticulosis

⇒ Small intestine

Angiodysplasia : thickening wall of intestine
 - Congo red stain
 Bact. overgrowth : stricture & diverticulae
 Celiac : villous atrophy

Infectious → all except giardiasis

lymphoma
 inflammation

⇒ PMC : edema
 hypoeosinophilic
 exudative
 skip areas

normal findings